

AN 1983:240354 BIOSIS

DN BA75:90354

TI INFLUENCE OF 6 METHOXY CUMARANON 2-ACETIC-ACID AND ITS DERIVATIVES ON THE PROCESS OF HUMORAL AND CELLULAR IMMUNOGENESIS.

AU ZABLOCKI B; KIERONSKA D; MARCZYK B; POZALSKA B; LAUK-PUCHALA B

CS DEP. IMMUNOL., INSTITUTE MICROBIOL., UNIV. LODZ, BANACHA 12/16, 90-237 LODZ.

SO ARCH IMMUNOL THER EXP, (1981 (RECD 1983)) 29 (6), 805-812.

CODEN: AITEAT. ISSN: 0004-069X.

FS BA; OLD

LA English

AB The influence of 6-metoxo-cumaranon-2-acetic acid and its 4 derivatives [6-hydroxy-cumaranon-2-acetic acid, 6-hydroxy-cumaranon-2-(N-4-carboxyphenyl-)acetamide, 6-hydroxy-cumaranon-2-(N-4-carboxymethylphenyl-) acetamide (HKCMF) and 6-hydroxy-cumaranon-2-(N-4-carboxy-3-hydroxy-phenyl-) acetamide (HKCHF)] on humoral and cellular immunogenesis was determined in mice. The study of immunosuppressive properties included: control of the level of peripheral blood lymphocytes, estimation of transplantation immunity, GvH [graft vs. host] reaction, PFC [plaque-forming cell] production for SRBCC [sheep erythrocyte] and LPS [lipopolysaccharide], and the determination of the number of cells possessing receptors for antigen, Fc fragment and complement. Blast transformation of lymphocytes stimulated with PHA [phytohemagglutinin] and the cytotoxic effect of sensitized lymphocytes were estimated. Results indicate immunosuppressive activity of acetamides HKCMF and HKCHF. They lowered the level of circulating lymphocytes, the number of cells possessing receptors for complement and they hindered PFC production for SRBC and LPS. Acetamide HKCHF weakened the cytotoxic activity of lymphocytes sensitized to alloantigens.

AB The influence of 6-metoxo-cumaranon-2-acetic acid and its 4 derivatives [6-hydroxy-cumaranon-2-acetic acid, 6-hydroxy-cumaranon-2-(N-4-carboxyphenyl-)acetamide, 6-hydroxy-cumaranon-2-(N-4-carboxymethylphenyl-) acetamide (HKCMF) and 6-hydroxy-cumaranon-2-(N-4-carboxy-3-hydroxy-phenyl-) acetamide (HKCHF)] on humoral and cellular immunogenesis was determined in mice. The study of immunosuppressive.

IT Miscellaneous Descriptors

MOUSE SHEEP LYMPHOCYTO TOXICITY PLAQUE FORMING CELL ERYTHROCYTE 6

HYDROXY CUMARANON-2-ACETIC-ACID 6 HYDROXY CUMARANON 2-N-4

CARBOXYPHENYL ACETAMIDE 6 HYDROXY CUMARANON-2 N-4

CARBOXYMETHYLPHENYL ACETAMIDE 6 HYDROXY CUMARANON 2-N-4

CARBOXY-3-HYDROXYPHENYL ACETAMIDE IMMUNOLOGIC-DRUG IMMUNO SUPPRESSANT

PHYTO HEM AGGLUTININ FC RECEPTOR COMPLEMENT GRAFT

VS. HOST REACTION

L8 ANSWER 2 OF 61 BIOTECHNO COPYRIGHT 2002 Elsevier Science B.V.

AN 1991:21339261 BIOTECHNO

TI Immunologically activated chloride channels involved in degranulation of rat mucosal mast cells

AU Romanin C.; Reinsprecht M.; Pecht I.; Schindler H.

CS Institute for Biophysics, University of Linz, A-4040 Linz, Austria.

SO EMBO Journal, (1991), 10/12 (3603-3608)

CODEN: EMJODG ISSN: 0261-4189

DT Journal; Article

CY United Kingdom

LA English

SL English

AB Crosslinking of type I Fc(.epsilon.) receptors (Fc(.epsilon.)RI) on the surface of basophils or mast cells initiates a cascade of processes leading to the secretion of inflammatory mediators. We report here a correlation between mediator secretion and the activation of Cl.sup.- channels in rat mucosal-type mast cells (line RBL-2H3). Stimulation of RBL cells by either IgE and antigen or by a monoclonal antibody specific for the Fc(.epsilon.)RI, resulted in the activation of Cl.sup.- ion channels as detected by the patch-clamp technique. Channel activation occurred slowly, within minutes after stimulation. The channel has a

slope conductance of 32 pS at potentials between 0 and -100 mV, and an increasing open-state probability with increasing depolarization. Activation of apparently the same Cl.sup.- channels could be mimicked without stimulation by isolating inside-out membrane patches in tyrode solution. Parallel inhibition of both Cl.sup.- channel activity and mediator secretion, as monitored by serotonin release, was observed by two compounds, the Cl.sup.- channel blocker 5-nitro-2-(3-phenylpropylamino) **benzoic acid** (NPPB) and the anti-allergic drug cromolyn. NPPB inhibited both the antigen-induced Cl.sup.- current and the serotonin release, where half-maximal inhibition occurred at similar doses, at 52 .mu.M and 77 .mu.M, respectively. The drug cromolyn, recently found to inhibit immunologically induced mediator secretion from RBL cells upon intracellular application, also blocks Cl.sup.- channels (IC.sub.50 = 15 .mu.M) when applied to the cytoplasmic side of an inside-out membrane patch. The observed Cl.sup.- channel activation upon immunological stimulation and the parallel inhibition of channel current and of serotonin release suggests a functional role for this Cl.sup.- channel in mediator secretion from the mast cells studied.

AB. . . channel activity and mediator secretion, as monitored by serotonin release, was observed by two compounds, the Cl.sup.- channel blocker 5-nitro-2-(3-phenylpropylamino) **benzoic acid** (NPPB) and the anti-allergic drug cromolyn. NPPB inhibited both the antigen-induced Cl.sup.- current and the serotonin release, where half-maximal inhibition. . .

CT *chloride channel; *immunostimulation; *serotonin release; **Fc receptor**; 5 nitro 2 (3 phenylpropylamino)**benzoic acid**; cromoglycate disodium; monoclonal antibody; animal cell; article; cross linking; degranulation; mast cell degranulation; mediator; mucosa cell; nonhuman; patch clamp; priority. . .

RN (5 nitro 2 (3 phenylpropylamino)**benzoic acid**)
107254-86-4; (cromoglycate disodium) 15826-37-6, 16110-51-3, 93356-79-7, 93356-84-4

L8 ANSWER 3 OF 61 CAPLUS COPYRIGHT 2002 ACS

AN 1995:644214 CAPLUS

DN 123:54051

TI The effect of a naphthalene derivative, TEI-6472, on histamine release and tyrosine phosphorylation in rat basophilic leukemia cells

AU Miyamoto, Hisashi; Matsui, Katsuhiko; Arai, Toshihiko

CS Dep. Microbiology, Meiji Coll. Pharmacy, Tokyo, 154, Japan

SO Int. J. Immunopharmacol. (1995), 17(5), 433-41

CODEN: IJIMDS; ISSN: 0192-0561

DT Journal

LA English

AB It is well known that rat basophilic leukemia cells (RBL-2H3) express high-affinity IgE receptors (Fc.epsilon.RI) and that the aggregation of these receptors causes the release of chem. mediators. When RBL-2H3 cells are sensitized with IgE and subsequently stimulated by an antigen, histamine release and the tyrosine phosphorylation of several proteins are obsd. Here, the authors examd. the effects of a synthetic naphthalene deriv., (7E)-N-(2-**carboxyphenyl**)-8-(2-naphthyl)-5,6-trans-5,6-methano-7-octenamide (TEI-6472), on the Fc.epsilon.RI-mediated histamine release from RBL-2H3 cells. Preincubation for 10 min with 100 .mu.M TEI-6472 caused inhibition of Fc.epsilon.RI-mediated histamine release from RBL-2H3 cells. Furthermore, Western blotting anal. using anti-phosphotyrosine antibody showed that Fc.epsilon.RI-mediated tyrosine phosphorylation of 78 and 92 kDa proteins in RBL-2H3 cells was also inhibited. Tyrosine phosphorylation of these 78 and 92 kDa proteins was not induced by direct activation of protein kinase C (PKC) by phorbol-12-myristate-13-acetate (PMA) and the calcium ionophore A23187. However, the inhibition of histamine release from TEI-6472-treated RBL-2H3 cells was restored by direct activation of PKC. Thus, tyrosine phosphorylation of the 78 and 92 kDa proteins in RBL-2H3 cells is involved in a signal transduction system for histamine secretion and the phosphorylation may occur upstream of PKC activation.

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IT Immunoglobulin receptors

Receptors

RL: BAC (Biological activity or effector, except adverse); BIOL (Biological study)

(Fc.epsilon.RI (IgE fragment **Fc receptor I**),

tyrosine phosphorylation upstream of protein kinase C activation is involved in Fc.epsilon.RI-mediated signaling for histamine secretion in basophils)

L8 ANSWER 4 OF 61 CAPLUS COPYRIGHT 2002 ACS

AN 1992:5109 CAPLUS

DN 116:5109

TI Immunologically activated chloride channels involved in degranulation of rat mucosal mast cells

AU Romanin, Christoph; Reinsprecht, Martin; Pecht, Israel; Schindler, Hansgeorg

CS Inst. Biophys., Univ. Linz, Linz, A-4040, Austria

SO EMBO J. (1991), 10(12), 3603-8

CODEN: EMJODG; ISSN: 0261-4189

DT Journal

LA English

AB Crosslinking of type I Fc.epsilon. receptors (Fc.epsilon.RI) on the surface of basophils or mast cells initiates a cascade of processes leading to the secretion of inflammatory mediators. The authors report here a correlation between mediator secretion and the activation of Cl⁻ channels in rat mucosal-type mast cells (line RBL-2H3). Stimulation of RBL cells by either IgE and antigen or by a monoclonal antibody specific for the Fc.epsilon.RI, resulted in the activation of Cl⁻ ion channels as detected by the patch-clamp technique. Channel has a slope conductance of 32 pS at potentials between 0 and -100 mV, and an increasing open-state probability with increasing depolarization. Activation of apparently the same Cl⁻ channels could be mimicked without stimulation by isolating inside-out membrane patches in Tyrode soln. Parallel inhibition of both Cl⁻ channel activity and mediator secretion, as monitored by serotonin release, was obsd. by two compds., the Cl⁻ channel blocker 5-nitro-2-(3-phenylpropylamino) **benzoic acid** (NPPB) and the anti-allergic drug cromolyn. NPPB inhibited both the antigen-induced Cl⁻ current and the serotonin release, where half-maximal inhibition occurred at similar doses, at 52 .mu.M and 77 .mu.M, resp. The drug cromolyn, recently found to inhibit immunol. induced mediator secretion from RBL cells upon intracellular application, also blocks Cl⁻ channels (IC50 = 15 .mu.M) when applied to the cytoplasmic side of an inside-out membrane patch. The obsd. Cl⁻ channel activation upon immunol. stimulation and the parallel inhibition of channel current and of serotonin release suggests a functional role for this Cl⁻ channel in mediator secretion from the mast cells studied.

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IT Receptors

(Fc.εpsilon.RI (IgE fragment **Fc receptor I**), chloride channel activation coupled to, in degranulation of mucosal mast cell)

L8 ANSWER 5 OF 61 DRUGU COPYRIGHT 2002 THOMSON DERWENT

AN 1994-28905 DRUGU P

TI Synthetic ligand to the retinoid X receptor (RXR) synergizes with either all-trans (T-) or 9-Cis (9-C-) retinoic acid (RA) to induce differentiation of myeloid leukemic cells.

AU Kerner B; Dawson M I; Faucett C; Koeffler H P

CS Univ.California

LO Los Angeles, Menlo Park, California, United States

SO Proc.Am.Assoc.Cancer Res. (35, 85 Meet., 274, 1994)

AV Department of Medicine, Cedars-Sinai Medical Center, UCLA, CA 90048, U.S.A.

LA English

DT Journal

FA AB; LA; CT

FS Literature

AN 1994-28905 DRUGU P

AB The synthetic ligand (4-(2-methyl-1-(5,6,7,8, -tetrahydro-5,5,8,8, tetramethyl 2-naphthalenyl) propen-1-yl)**benzoic acid**, (SRI-11217) to the retinoid X receptor (RXR) synergized with either all trans or 9-cis retinoic acid (RA) to induce differentiation of myeloid leukemic cells. SRI-11217 alone, had almost no activity. SRI-11217 had no effect on expression of the low affinity **Fc receptor** for IgE (CD23). Combinations of SRI-11217 with either T-RA or 9-C-RA synergistically increased expression of CD23. These results show that a ligand to the RXR synergize with a ligand to retinoic acid receptor (RAR). Furthermore, the data suggest that amplification of the hormone response to T-RA is possible with ligands specific for RXR. (conference abstract).

ABEX SRI-11217 alone, had almost no activity. There was a synergistic effect of low concentrations of either 9-C-RA or T-RA with SRI-11217, as measured by inhibition of clonal proliferation and induction of differentiation of HL-60 cells. The expression of the low affinity **Fc receptor** for IgE (CD23), which is up-regulated during RA-induced myeloid differentiation was examined. Treatment of HL-60 cells with either T-RA or 9-C-RA (10 nM) for 5 days moderately increased the expression of CD23 antigen. In contrast, SRI-11217 (1 μM-10 nM) had no effect on expression of this receptor. Combination of SRI-11217 (1 μM) with either T-RA or 9-C-RA synergistically increased

expression of CD23. (KJ)

AB The synthetic ligand (4-(2-methyl-1-(5,6,7,8, -tetrahydro-5,5,8,8, tetramethyl 2-naphthalenyl) propen-1-yl)**benzoic acid**, (SR-11217) to the retinoid X receptor (RXR) synergized with either all trans or 9-cis retinoic acid (RA) to induce differentiation. . . of myeloid leukemic cells. SRI-11217 alone, had almost no activity. SRI-11217 had no effect on expression of the low affinity **Fc receptor** for IgE (CD23). Combinations of SRI-11217 with either T-RA or 9-C-RA synergistically increased expression of CD23. These results show that. . .

ABEX. . . as measured by inhibition of clonal proliferation and induction of differentiation of HL-60 cells. The expression of the low affinity **Fc receptor** for IgE (CD23), which is up-regulated during RA-induced myeloid differentiation was examined. Treatment of HL-60 cells with either T-RA or. . .

CT MYELOID *FT; IN-VITRO *FT; TUMOR-CELL *FT; LEUKEMIA *FT; COMB. *FT; **FC-RECEPTOR** *FT; EXPRESSION *FT; CD23 *FT; CYTOSTATIC *FT; TISSUE-CULTURE *FT

CT MYELOID *FT; IN-VITRO *FT; TUMOR-CELL *FT; LEUKEMIA *FT; COMB. *FT; **FC-RECEPTOR** *FT; EXPRESSION *FT; CD23 *FT; CYTOSTATIC *FT; TISSUE-CULTURE *FT

L8 ANSWER 6 OF 61 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.

AN 2001225012 EMBASE

TI Cytotoxicity testing of wound-dressing materials.

AU Sahlin H.; Nygren H.

CS H. Sahlin, Applied Cell Biology, Department of Anatomy, Goteborg University, P.O. Box 420, 405 30 Gothenburg, Sweden

SO ATLA Alternatives to Laboratory Animals, (2001) 29/3 (269-275).
Refs: 13
ISSN: 0261-1929 CODEN: AALADQ

CY United Kingdom

DT Journal; Conference Article

FS 052 Toxicology

LA English

SL English

AB A method was developed for testing the cytotoxicity of various bandage-like wound dressings and gel wound dressings. In this method, the ability of human polymorphonuclear neutrophils (PMNs) to initiate a respiratory burst after exposure to the various wound dressings is used as a marker of cytotoxicity. Luminol-amplified chemiluminescence stimulated with opsonised zymosan or phorbol 12-myristate 13-acetate (PMA) is used to measure the degree of activation of the respiratory burst, i.e. the NADPH oxidase activity, after exposure to wound dressings. Opsonised zymosan (material from yeast cell walls) is a phagocytic stimulus that activates the NADPH oxidase by binding to Fe-receptors and complement receptors, and functions as an artificial bacterium, whereas PMA activates the NADPH oxidase by direct activation of protein kinase C. NADPH oxidase activity was inhibited by several wound dressings. The down-regulation of the respiratory burst is detrimental to the bactericidal effect of PMNs, and can be used as a marker for the cytotoxicity of wound-dressing materials.

CT Medical Descriptors:
*immunotoxicity: . . . paper
priority journal
*gelling agent: TO, drug toxicity
acemannan: TO, drug toxicity
zymosan
reduced nicotinamide adenine dinucleotide dehydrogenase: EC, endogenous compound
phorbol 13 acetate 12 myristate
Fc receptor: EC, endogenous compound
complement receptor: EC, endogenous compound
carbomer: TO, drug toxicity
povidone: TO, drug toxicity
alginic acid: TO, drug toxicity
carboxymethylcellulose: TO, drug toxicity
guar gum: TO, drug toxicity

pectin: TO, drug toxicity
xanthan: TO, drug toxicity
collagen: TO, drug toxicity
sorbate potassium: TO, drug toxicity

benzoic acid: TO, drug toxicity
sodium metabisulfite: TO, drug toxicity
propylene glycol: TO, drug toxicity
methyl paraben: TO, drug toxicity
germall 115: TO, drug toxicity
propyl.

RN. . . 29894-36-8, 9005-32-7, 9005-38-3; (carboxymethylcellulose)
8050-38-2, 9000-11-7, 9004-32-4, 9050-04-8; (guar gum) 9000-30-0; (pectin)
9000-69-5; (xanthan) 11138-66-2; (collagen) 9007-34-5; (sorbate potassium)
24634-61-5; (**benzoic acid**) 532-32-1, 582-25-2,
65-85-0, 766-76-7; (sodium metabisulfite) 7681-57-4, 7757-74-6; (propylene
glycol) 57-55-6; (methyl paraben) 99-76-3; (germall 115) 39236-46-9;
(propyl paraben) 94-13-3

L8 ANSWER 7 OF 61 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.

AN 1999108984 EMBASE

TI 4-Trifluoromethyl derivatives of salicylate, triflusal and its main
metabolite 2-hydroxy-4-trifluoromethylbenzoic acid, are potent inhibitors
of nuclear factor .kappa.B activation.

AU Bayon Y.; Alonso A.; Crespo M.S.

CS M.S. Crespo, Inst. Biologia y Genetica Molecular, CSIC, Facultad de
Medicina, 47005-Valladolid, Spain. mscres@ibgm.uva.es

SO British Journal of Pharmacology, (1999) 126/6 (1359-1366).

Refs: 40

ISSN: 0007-1188 CODEN: BJPCBM

CY United Kingdom

DT Journal; Article

FS 029 Clinical Biochemistry

030 Pharmacology

037 Drug Literature Index

LA English

SL English

AB 1. The effect of two derivatives of salicylate, 2-hydroxy-4-
trifluoromethylbenzoic acid (HTB) and 2-acetoxy-4-trifluoromethylbenzoic
acid (triflusal), on the activation of NF-.kappa.B elicited by tumour
necrosis factor-.alpha. (TNF-.alpha.) on human umbilical vein endothelial
cells (HUVEC) was tested. 2. The expression of the mRNA of vascular cell
adhesion molecule-1 (VCAM-1) was studied as an example of a gene the
expression of which is regulated by NF-.kappa.B. To extend these findings
to other systems, the induction of nitric oxide synthase in rat adherent
peritoneal macrophages was studied. 3. Both HTB and triflusal were more
potent than aspirin or salicylate as inhibitors of the nuclear
translocation of NF-.kappa.B. The calculation of the IC50 values showed
.simeq. 2 mM for HTB, 4 mM for aspirin and > 4 mM for salicylate. 4.
Comparison of the potency of these compounds on VCAM-1 mRNA expression
showed complete inhibition by both triflusal and HTB at a concentration of
4 mM whereas aspirin and salicylate produced only 36-43% inhibition at the
same concentration. 5. Inhibition of NF-.kappa.B activation was also
observed in rat peritoneal macrophages stimulated via their receptors for
the Fc portion of the antibody molecule with IgG/ovalbumin immune
complexes. This was accompanied by a dose-dependent inhibition of nitrite
production by the L-arginine pathway via iNOS. IC50 values for this effect
were 1.13 .+-. 0.12 mM (triflusal), 1.84 .+-. 0.34 (HTB), 6.08 .+-. 1.53
mM (aspirin) and 9.16 .+-. 1.9 mM (salicylate). 6. These data indicate
that the incorporation of a 4-trifluoromethyl group to the salicylate
molecule strongly enhances its inhibitory effect on NF-.kappa.B
activation, VCAM-1 mRNA expression and iNOS induction, irrespective of the
presence of the acetyl moiety involved in the inhibition of
cyclo-oxygenase.

CT Medical Descriptors:

*transcription . . .

DV, drug development

*salicylic acid derivative: PD, pharmacology

*triflusal: CM, drug comparison
 *triflusal: DV, drug development
 *triflusal: PD, pharmacology
 *immunoglobulin enhancer binding protein: EC, endogenous compound
 benzoic acid derivative: CM, drug comparison
 benzoic acid derivative: DV, drug development
 benzoic acid derivative: PD, pharmacology
 tumor necrosis factor alpha
 messenger RNA: EC, endogenous compound
 vascular cell adhesion molecule 1: EC, endogenous compound
 nitric oxide synthase: EC, endogenous compound
 salicylic acid: CM, drug comparison
 acetylsalicylic acid: CM, drug comparison
 Fc receptor: EC, endogenous compound
 nitrite: EC, endogenous compound

L8 ANSWER 8 OF 61 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
 AN 95173813 EMBASE
 DN 1995173813
 TI The effect of a naphthalene derivative, TEI-6472, on histamine release and tyrosine phosphorylation in rat basophilic leukemia cells.
 AU Miyamoto H.; Matsui K.; Arai T.
 CS Department of Microbiology, Meiji College of Pharmacy, 1-35-23 Nozawa, Setagaya-ku, Tokyo 154, Japan
 SO International Journal of Immunopharmacology, (1995) 17/5 (433-441).
 ISSN: 0192-0561 CODEN: IJIMDS
 CY United Kingdom
 DT Journal; Article
 FS 016 Cancer
 025 Hematology
 026 Immunology, Serology and Transplantation
 030 Pharmacology
 037 Drug Literature Index
 LA English
 SL English
 AB It is well known that rat basophilic leukemia cells (RBL-2H3) express high-affinity IgE receptors (Fc.epsilon.RIU) and that the aggregation of these receptors causes the release of chemical mediators. When RBL-2H3 cells are sensitized with IgE antibody and subsequently stimulated by an antigen, significant histamine release and the tyrosine phosphorylation of several proteins are observed. In this study, we examined the effects of a synthetic naphthalene derivative, (7E)-N-(2-carboxyphenyl)-8-(2-naphthyl)-5,6-trans-5,6-methano-7-octenamide (TEI-6472), on the Fc.epsilon.RI-mediated histamine release from RBL-2H3 cells. Preincubation for 10 min with 100 .mu.M TEI-6472 caused significant inhibition of Fc.epsilon.RI-mediated histamine release from RBL-2H3 cells. Furthermore, Western blotting analysis using anti-phosphotyrosine antibody showed that Fc.epsilon.RI-mediated tyrosine phosphorylation of 78 and 92 kDa proteins in RBL-2H3 cells was also significantly inhibited. Tyrosine phosphorylation of these 78 and 92 kDa proteins was not induced by direct activation of protein kinase C (PKC) by phorbol-12-myristate-13-acetate (PMA) and the calcium ionophore A23187. However, the inhibition of histamine release from TEI-6472-treated RBL-2H3 cells was restored by direct activation of PKC. Taken together, these results suggest that tyrosine phosphorylation of the 78 and 92 kDa proteins in RBL-2H3 cells is involved in a signal transduction system for histamine secretion, and that these tyrosine phosphorylations may occur upstream of PKC activation.
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 CT Medical Descriptors:
 *histamine release
 *mast cell leukemia
 *protein phosphorylation

*signal transduction
animal cell
article
nonhuman
priority journal
rat

Fc receptor

*histamine: EC, endogenous compound
*immunoglobulin e: EC, endogenous compound
*immunomodulating agent: PD, pharmacology
*naphthalene derivative: PD, pharmacology
*phorbol ester
*protein kinase c: EC, endogenous compound
tei. . .

L8 ANSWER 9 OF 61 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.

AN 91339955 EMBASE

DN 1991339955

TI Immunologically activated chloride channels involved in degranulation of
rat mucosal mast cells.

AU Romanin C.; Reinsprecht M.; Pecht I.; Schindler H.

CS Institute for Biophysics, University of Linz, A-4040 Linz, Austria

SO EMBO Journal, (1991) 10/12 (3603-3608).

ISSN: 0261-4189 CODEN: EMJODG

CY United Kingdom

DT Journal; Article

FS 026 Immunology, Serology and Transplantation

LA English

SL English

AB Crosslinking of type I Fc(.epsilon.) receptors (Fc(.epsilon.)RI) on the
surface of basophils or mast cells initiates a cascade of processes
leading to the secretion of inflammatory mediators. We report here a
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in rat mucosal-type mast cells (line RBL-2H3). Stimulation of RBL cells by
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detected by the patch-clamp technique. Channel activation occurred slowly,
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also blocks Cl⁻ channels (IC50 = 15 .mu.M) when applied to the cytoplasmic
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upon immunological stimulation and the parallel inhibition of channel
current and of serotonin release suggests a functional role for this Cl⁻
channel in mediator secretion from the mast cells studied.

AB . . . channel activity and mediator secretion, as monitored by
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and the anti-allergic drug cromolyn. NPPB inhibited both the
antigen-induced Cl⁻ current and the serotonin release, where half-maximal
inhibition. . .

CT Medical Descriptors:

*chloride channel
*immunostimulation
*serotonin release
animal cell
article

cross linking
degranulation
mast cell degranulation
mediator
mucosa cell
nonhuman
patch clamp
priority journal
rat
secretion
stimulation

Fc receptor

5 nitro 2 (3 phenylpropylamino)benzoic acid

cromoglycate disodium
monoclonal antibody

RN (5 nitro 2 (3 phenylpropylamino)benzoic acid)
107254-86-4; (cromoglycate disodium) 15826-37-6, 16110-51-3, 93356-79-7,
93356-84-4

L8 ANSWER 10 OF 61 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.

AN 77121160 EMBASE

DN 1977121160

TI Receptors for antibody opsonic adherence on the eosinophils of guinea pigs.

AU Butterworth A.E.; Coombs R.R.A.; Gurner B.W.; Wilson A.B.

CS Div. Immunol., Dept. Pathol., Univ. Cambridge, United Kingdom

SO International Archives of Allergy and Applied Immunology, (1976) 51/3
(368-377).

CODEN: IAAAAM

DT Journal

FS 026 Immunology, Serology and Transplantation

025 Hematology

LA English

AB Eosinophils have recently been implicated in antibody dependent cell mediated damage to schistosomula. Because of this, eosinophils of the guinea pig have been examined for surface receptors capable of giving antibody opsonic adherence; a rosetting reaction has been used. The eosinophils were shown to possess Fc receptors for homologous immunoglobulin. No selective difference between IgG1 and IgG2 was observed. In marked contrast to macrophages, guinea pig eosinophils failed to show opsonic adherence to red cells sensitized to a comparable degree with rabbit antibody. With red cell antibodies made in the pig, however, the reciprocal situation held, namely opsonic adherence was stronger with eosinophils than with macrophages.

CT Medical Descriptors:

***4 (1 imidazolylmethyl)benzoic acid**

*cytolysis

*eosinophil

*humoral immunity

in vitro study

theoretical study

guinea pig

***Fc receptor**

L8 ANSWER 11 OF 61 MEDLINE

AN 89008870 MEDLINE

DN 89008870 PubMed ID: 3049672

TI Studies on the molecular mechanisms of human **Fc receptor**-mediated phagocytosis. Amplification of ingestion is dependent on the generation of reactive oxygen metabolites and is deficient in polymorphonuclear leukocytes from patients with chronic granulomatous disease.

AU Gresham H D; McGarr J A; Shackelford P G; Brown E J

CS Department of Medicine, Washington University School of Medicine, St. Louis, Missouri 63110.

NC AI-19350 (NIAID)

AI-23790 (NIAID)

GM-38330 (NIGMS)
SO JOURNAL OF CLINICAL INVESTIGATION, (1988 Oct) 82 (4) 1192-201.
Journal code: HS7; 7802877. ISSN: 0021-9738.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Abridged Index Medicus Journals; Priority Journals
EM 198811
ED Entered STN: 19900308
Last Updated on STN: 19990129
Entered Medline: 19881115
AB Human PMN and monocytes both possess a mechanism for amplifying **Fc receptor**-mediated phagocytic function, which is dependent on activation of the respiratory burst. The pathway for augmentation of phagocytosis requires superoxide anion, hydrogen peroxide, and lactoferrin and is independent of the hydrogen peroxide-MPO-halide system. In neither cell type is this mechanism induced upon exposure to the opsonized target. PMN require an additional signal for stimulation of the respiratory burst; this is not true of monocytes. On the other hand, monocytes require an exogenous source of lactoferrin in order to activate this pathway for enhanced ingestion. The dependence of this pathway for both PMN and monocytes on superoxide anion, hydrogen peroxide, and cell-bound lactoferrin is consistent with a role for locally generated reactive oxygen metabolites, possibly hydroxyl radicals, in phagocytosis amplification. Patients with chronic granulomatous disease, who are genetically deficient in the ability to activate the respiratory burst, are unable to amplify **Fc receptor**-mediated phagocytosis. Thus, these patients may have a previously unrecognized defect in the recruitment of phagocytic function at inflammatory sites.
TI Studies on the molecular mechanisms of human **Fc receptor**-mediated phagocytosis. Amplification of ingestion is dependent on the generation of reactive oxygen metabolites and is deficient in polymorphonuclear leukocytes from.
AB Human PMN and monocytes both possess a mechanism for amplifying **Fc receptor**-mediated phagocytic function, which is dependent on activation of the respiratory burst. The pathway for augmentation of phagocytosis requires superoxide anion, . . . with chronic granulomatous disease, who are genetically deficient in the ability to activate the respiratory burst, are unable to amplify **Fc receptor**-mediated phagocytosis. Thus, these patients may have a previously unrecognized defect in the recruitment of phagocytic function at inflammatory sites.
CT . . . Human; Male; Support, Non-U.S. Gov't; Support, U.S. Gov't, P.H.S.
Amphotericin B: AI, antagonists & inhibitors
Amphotericin B: PD, pharmacology
Benzoates
Benzoic Acid
Biological Factors: AI, antagonists & inhibitors
Biological Factors: PD, pharmacology
Catalase
Cytokines
Free Radicals
*Granulomatous Disease, Chronic: BL, . . .
RN 1397-89-3 (Amphotericin B); 37558-16-0 (Phorbol 12,13-Dibutyrate); 65-85-0 (Benzoic Acid); 9010-72-4 (Zymosan)
L8 ANSWER 12 OF 61 TOXCENTER COPYRIGHT 2002 ACS
AN 1989:4894 TOXCENTER
DN 89008870 PubMed ID: 3049672
TI Studies on the molecular mechanisms of human **Fc receptor**-mediated phagocytosis. Amplification of ingestion is dependent on the generation of reactive oxygen metabolites and is deficient in polymorphonuclear leukocytes from patients with chronic granulomatous disease
AU Gresham H D; McGarr J A; Shackelford P G; Brown E J
CS Department of Medicine, Washington University School of Medicine, St. Louis, Missouri 63110

NC AI-19350 (NIAID)
 AI-23790 (NIAID)
 GM-38330 (NIGMS)
 SO JOURNAL OF CLINICAL INVESTIGATION, (1988 Oct) 82 (4) 1192-201.
 Journal Code: HS7; 7802877. ISSN: 0021-9738.
 CY United States
 DT Journal; Article; (JOURNAL ARTICLE)
 FS MEDLINE
 OS MEDLINE 89008870
 LA English
 ED Entered STN: 20011116
 Last Updated on STN: 20011116
 AN 1989:4894 TOXCENTER
 AB Human PMN and monocytes both possess a mechanism for amplifying **Fc receptor**-mediated phagocytic function, which is dependent on activation of the respiratory burst. The pathway for augmentation of phagocytosis requires superoxide anion, hydrogen peroxide, and lactoferrin and is independent of the hydrogen peroxide-MPO-halide system. In neither cell type is this mechanism induced upon exposure to the opsonized target. PMN require an additional signal for stimulation of the respiratory burst; this is not true of monocytes. On the other hand, monocytes require an exogenous source of lactoferrin in order to activate this pathway for enhanced ingestion. The dependence of this pathway for both PMN and monocytes on superoxide anion, hydrogen peroxide, and cell-bound lactoferrin is consistent with a role for locally generated reactive oxygen metabolites, possibly hydroxyl radicals, in phagocytosis amplification. Patients with chronic granulomatous disease, who are genetically deficient in the ability to activate the respiratory burst, are unable to amplify **Fc receptor**-mediated phagocytosis. Thus, these patients may have a previously unrecognized defect in the recruitment of phagocytic function at inflammatory sites.
 TI Studies on the molecular mechanisms of human **Fc receptor**-mediated phagocytosis. Amplification of ingestion is dependent on the generation of reactive oxygen metabolites and is deficient in polymorphonuclear leukocytes from. . .
 AB Human PMN and monocytes both possess a mechanism for amplifying **Fc receptor**-mediated phagocytic function, which is dependent on activation of the respiratory burst. The pathway for augmentation of phagocytosis requires superoxide anion, . . . with chronic granulomatous disease, who are genetically deficient in the ability to activate the respiratory burst, are unable to amplify **Fc receptor**-mediated phagocytosis. Thus, these patients may have a previously unrecognized defect in the recruitment of phagocytic function at inflammatory sites.
 CT . . . Human; Male; Support, Non-U.S. Gov't; Support, U.S. Gov't, P.H.S.
 Amphotericin B: AI, antagonists & inhibitors
 Amphotericin B: PD, pharmacology
 Benzoates
 Benzoic Acid
 Biological Factors: AI, antagonists & inhibitors
 Biological Factors: PD, pharmacology
 Catalase
 Cytokines
 Free Radicals
 *Granulomatous Disease, Chronic: BL, blood
 Granulomatous. . .
 RN 1397-89-3 (Amphotericin B)
 37558-16-0 (Phorbol 12,13-Dibutyrate)
 65-85-0 (**Benzoic Acid**)
 9010-72-4 (Zymosan)
 L8 ANSWER 13 OF 61 TOXCENTER COPYRIGHT 2002 ACS
 AN 1983:75994 TOXCENTER
 CP Copyright 2002.BIOSIS
 DN BA75:90354
 TI INFLUENCE OF 6 METHOXY CUMARANON 2-ACETIC-ACID AND ITS DERIVATIVES ON THE PROCESS OF HUMORAL AND CELLULAR IMMUNOGENESIS

AU ZABLOCKI B; KIERONSKA D; MARCZYK B; POZALSKA B; LAUK-PUCHALA B
 CS DEP. IMMUNOL., INSTITUTE MICROBIOL., UNIV. LODZ, BANACHA 12/16, 90-237
 LODZ.
 SO ARCH IMMUNOL THER EXP, (1981 (RECD 1983)) 29 (6), 805-812
 CODEN: AITEAT. ISSN: 0004-069X.
 FS BIOSIS
 OS BIOSIS 1983:240354
 LA English
 ED Entered STN: 20011116
 Last Updated on STN: 20011116
 AN 1983:75994 TOXCENTER
 CP Copyright 2002 BIOSIS
 AB The influence of 6-metoxy-cumaranon-2-acetic acid and its 4 derivatives
 [6-hydroxy-cumaranon-2-acetic acid, 6-hydroxy-cumaranon-2-(N-4-
carboxyphenyl-)acetamide, 6-hydroxy-cumaranon-2-(N-4-
 carboxymethylphenyl-) acetamide (HKCMF) and 6-hydroxy-cumaranon-2-(N-4-
 carboxy-3-hydroxy-phenyl-) acetamide (HKCHF)] on humoral and cellular
 immunogenesis was determined in mice. The study of immunosuppressive
 properties included: control of the level of peripheral blood lymphocytes,
 estimation of transplantation immunity, GvH [graft vs. host] reaction, PFC
 [plaque-forming cell] production for SRBCC [sheep erythrocyte] and LPS
 [lipopolysaccharide], and the determination of the number of cells possessing
 receptors for antigen, Fc fragment and complement. Blast transformation
 of lymphocytes stimulated with PHA [phytohemagglutinin] and the cytotoxic
 effect of sensitized lymphocytes were estimated. Results indicate
 immunosuppressive activity of acetamides HKCMF and HKCHF. They lowered
 the level of circulating lymphocytes, the number of cells possessing
 receptors for complement and they hindered PFC production for SRBC and
 LPS. Acetamide HKCHF weakened the cytotoxic activity of lymphocytes
 sensitized to alloantigens.
 AB The influence of 6-metoxy-cumaranon-2-acetic acid and its 4 derivatives
 [6-hydroxy-cumaranon-2-acetic acid, 6-hydroxy-cumaranon-2-(N-4-
carboxyphenyl-)acetamide, 6-hydroxy-cumaranon-2-(N-4-
 carboxymethylphenyl-) acetamide (HKCMF) and 6-hydroxy-cumaranon-2-(N-4-
 carboxy-3-hydroxy-phenyl-) acetamide (HKCHF)] on humoral and cellular
 immunogenesis was determined in mice. The study of immunosuppressive.
 ST Miscellaneous Descriptors
 MOUSE SHEEP LYMPHOCYTO TOXICITY PLAQUE FORMING CELL ERYTHROCYTE 6
 HYDROXY CUMARANON-2-ACETIC-ACID 6 HYDROXY CUMARANON 2-N-4
CARBOXYPHENYL ACETAMIDE 6 HYDROXY CUMARANON-2 N-4
 CARBOXYMETHYLPHENYL ACETAMIDE 6 HYDROXY CUMARANON 2-N-4
 CARBOXY-3-HYDROXYPHENYL ACETAMIDE IMMUNOLOGIC-DRUG IMMUNO SUPPRESSANT
 PHYTO HEM AGGLUTININ **FC RECEPTOR** COMPLEMENT GRAFT
 VS. HOST REACTION
 L8 ANSWER 14 OF 61 USPATFULL
 AN 2002:92268 USPATFULL
 TI Human G-protein Chemokine Receptor HDGNR10
 IN Rosen, Craig A., Laytonsville, MD, UNITED STATES
 Roschke, Viktor, Rockville, MD, UNITED STATES
 Li, Yi, Sunnyvale, CA, UNITED STATES
 Ruben, Steven M., Olney, MD, UNITED STATES
 PI US 2002048786 A1 20020425
 AI US 2001-779879 A1 20010209 (9)
 PRAI US 2000-181258P 20000209 (60)
 US 2000-187999P 20000309 (60)
 US 2000-234336P 20000922 (60)
 DT Utility
 FS APPLICATION
 LREP STERNE, KESSLER, GOLDSTEIN & FOX PLLC, 1100 NEW YORK AVENUE, N.W., SUITE
 600, WASHINGTON, DC, 20005-3934
 CLMN Number of Claims: 61
 ECL Exemplary Claim: 1
 DRWN 4 Drawing Page(s)
 LN.CNT 17969
 AB The present invention relates to a novel human protein called Human

G-protein Chemokine Receptor (CCR5) HDGNR10, and isolated polynucleotides encoding this protein. The invention is also directed to human antibodies that bind Human G-protein Chemokine Receptor (CCR5) HDGNR10 and to polynucleotides encoding those antibodies. Also provided are vectors, host cells, antibodies, and recombinant methods for producing Human G-protein Chemokine Receptor (CCR5) HDGNR10 and human anti-Human G-protein Chemokine Receptor (CCR5) HDGNR10 antibodies. The invention further relates to diagnostic and therapeutic methods useful for diagnosing and treating diseases, disorders, and/or conditions related to this novel human protein and these novel human antibodies.

DETD . . . that may be used according to the methods of the invention include, but are not limited to, glutamyl derivatives of **benzoic acid** mustard alkylating agent, phosphate derivatives of etoposide or mitomycin C, cytosine arabinoside, daunorubisin, and phenoxylacetamide derivatives of doxorubicin.

DETD . . . Three major families of cell surface antigens can be identified on monocytes: adhesion molecules, molecules involved in antigen presentation, and **Fc receptor**. Modulation of the expression of MHC class II antigens and other costimulatory molecules, such as B7 and ICAM-1, may result. . .

L8 ANSWER 15 OF 61 USPATFULL

AN 2002:88256 USPATFULL

TI Recombinant alphavirus particles

IN Dubensky, Jr., Thomas W., Rancho Sante Fe, CA, United States

Polo, John M., San Diego, CA, United States

Ibanez, Carlos E., San Diego, CA, United States

Driver, David A., San Diego, CA, United States

PA Chiron Corporation, Emeryville, CA, United States (U.S. corporation)

PI US 6376236 B1 20020423

AI US 1999-236140 19990122 (9)

RLI Division of Ser. No. US 1995-404796, filed on 15 Mar 1995, now patented, Pat. No. US 6015686 Continuation-in-part of Ser. No. US 1995-376184, filed on 18 Jan 1995, now abandoned Continuation-in-part of Ser. No. US 1994-348472, filed on 30 Nov 1994, now abandoned Continuation-in-part of Ser. No. US 1994-198450, filed on 18 Feb 1994, now abandoned Continuation-in-part of Ser. No. US 1993-122791, filed on 15 Sep 1993, now abandoned

DT Utility

FS GRANTED

EXNAM Primary Examiner: Brusca, John S.

LREP McMasters, David D., Dollard, Anne S., Blackburn, Robert P.

CLMN Number of Claims: 11

ECL Exemplary Claim: 1

DRWN 37 Drawing Figure(s); 30 Drawing Page(s)

LN.CNT 9308

AB Disclosed are recombinant alphavirus particles comprising a) an alphavirus vector construct which directs the expression of a heterologous nucleic acid molecule; b) a capsid protein; and c) an envelope glycoprotein from a virus different from said alphavirus vector.

DETD . . . 89:33, 1992); carboxypeptidase G2, which will cleave the glutamic acid from para-N-bis (2-chloroethyl) aminobenzoyl glutamic acid, thereby creating a toxic **benzoic acid** mustard; and Penicillin-V amidase, which will convert phenoxylacetamide derivatives of doxorubicin and melphalan to toxic compounds (see generally, Vruthula et. . .

DETD . . . proteins that recognize Fc portions of antibodies. Monoclonal antibodies which recognize only preselected target cells are then bound to such **Fc receptor**-bearing alphavirus vector particles, such that the vector particles bind to and infect only those preselected target cells (for example, tumor. . .

L8 ANSWER 16 OF 61 USPATFULL

AN 2002:85173 USPATFULL

TI IL-17 receptor like molecules and uses thereof
IN Jing, Shugian, Thousand Oaks, CA, UNITED STATES
PI US 2002045213 A1 20020418
AI US 2001-809567 A1 20010315 (9)
RLI Continuation-in-part of Ser. No. US 2000-724460, filed on 28 Nov 2000,
PENDING
PRAI US 2000-189816P 20000316 (60)
DT Utility
FS APPLICATION
LREP MARSHALL, O'TOOLE, GERSTEIN, MURRAY & BORUN, 6300 SEARS TOWER, 233 SOUTH
WACKER DRIVE, CHICAGO, IL, 60606-6402
CLMN Number of Claims: 71
ECL Exemplary Claim: 1
DRWN 4 Drawing Page(s)
LN.CNT 4685

AB Novel IL-17 receptor like polypeptides and nucleic acid molecules
encoding the same. The invention also provides vectors, host cells,
agonists and antagonists (including selective binding agents), and
methods for producing IL-17 receptor like polypeptides. Also provided
for are methods for the treatment, diagnosis, amelioration, or
prevention of diseases with IL-17 receptor like polypeptides.

DETD . . . (1989). When constructed together with a therapeutic protein,
an Fc domain can provide longer half-life or incorporate such functions
as **Fc receptor** binding, protein A binding,
complement fixation and perhaps even placental transfer. Id. Table II
summarizes the use of certain Fc. . .

DETD . . . agents, hydrophilic polymers (such as polyvinylpyrrolidone),
low molecular weight polypeptides, salt-forming counterions (such as
sodium), preservatives (such as benzalkonium chloride, **benzoic
acid**, salicylic acid, thimerosal, phenethyl alcohol,
methylparaben, propylparaben, chlorhexidine, sorbic acid or hydrogen
peroxide), solvents (such as glycerin, propylene glycol or. . .

L8 ANSWER 17 OF 61 USPATFULL

AN 2002:84902 USPATFULL

TI Nucleic acids, proteins and antibodies

IN Rosen, Craig A., Laytonsville, MD, UNITED STATES

Ruben, Steven M., Olney, MD, UNITED STATES

PI US 2002044941 A1 20020418

AI US 2001-925302 A1 20010810 (9)

RLI Continuation-in-part of Ser. No. WO 2000-US5918, filed on 8 Mar 2000,
UNKNOWN

PRAI US 1999-124270P 19990312 (60)

DT Utility

FS APPLICATION

LREP HUMAN GENOME SCIENCES INC, 9410 KEY WEST AVENUE, ROCKVILLE, MD, 20850

CLMN Number of Claims: 23

ECL Exemplary Claim: 1

DRWN No Drawings

LN.CNT 21121

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The present invention relates to novel lung cancer related
polynucleotides, the polypeptides encoded by these polynucleotides
herein collectively referred to as "lung cancer antigens," and
antibodies that immunospecifically bind these polypeptides, and the use
of such lung cancer polynucleotides, antigens, and antibodies for
detecting, treating, preventing and/or prognosing disorders of the lung,
including, but not limited to, the presence of lung cancer and lung
cancer metastases. More specifically, isolated lung cancer nucleic acid
molecules are provided encoding novel lung cancer polypeptides. Novel
lung cancer polypeptides and antibodies that bind to these polypeptides
are provided. Also provided are vectors, host cells, and recombinant and
synthetic methods for producing human lung cancer polynucleotides,
polypeptides, and/or antibodies. The invention further relates to
diagnostic and therapeutic methods useful for diagnosing, treating,
preventing and/or prognosing disorders related to the lung, including

lung cancer, and therapeutic methods for treating such disorders. The invention further relates to screening methods for identifying agonists and antagonists of polynucleotides and polypeptides of the invention. The invention further relates to methods and/or compositions for inhibiting or promoting the production and/or function of the polypeptides of the invention.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

SUMM . . . gi|182474 51 293 98 98 HAPBV45

>pir|JL0118|JL0118 Fc gamma (IgG) receptor IIa
precursor - human >sp|P12318|FCGA_HUMAN
LOW AFFINITY IMMUNOGLOBULIN GAMMA
FC RECEPTOR II-A PRECURSOR (FC-GAMMA
RII-A) (FCRII-A) (IGG FC RECEPTOR

II-A)

(CD32)

5 540125 cyclin H [Homo sapiens] >gi|532561 cyclin H
gi|536920 80 1099 95 95 HFPCA09

[Homo sapiens] >pir|I38731|I38731 cyclin.

SUMM . . . that may be used according to the methods of the invention
include, but are not limited to, glutamyl derivatives of **benzoic
acid** mustard alkylating agent, phosphate derivatives of
etoposide or mitomycin C, cytosine arabinoside, daunorubisin, and
phenoxacetamide derivatives of doxorubicin.

SUMM . . . Clin. Invest. 79:1440-1446 (1987)); anticollagenase-serum;
alpha2-antiplasmin (Holmes et al., J. Biol. Chem. 262(4):1659-1664
(1987)); Bisantrene (National Cancer Institute); Lobenzarit disodium
(N-(2)-**carboxyphenyl**-4- chloroanthronilic acid disodium or
"CCA"; Takeuchi et al., Agents Actions 36:312-316, 1992); Thalidomide;
Angostatic steroid; AGM-1470; carboxynaminolmidazole; and
metalloproteinase inhibitors.

SUMM . . . that may be used according to the methods of the invention
include, but are not limited to, glutamyl derivatives of **benzoic
acid** mustard alkylating agent, phosphate derivatives of
etoposide or mitomycin C, cytosine arabinoside, daunorubisin, and
phenoxacetamide derivatives of doxorubicin. Drug Screening

DETD . . . Three major families of cell surface antigens can be identified
on monocytes: adhesion molecules, molecules involved in antigen
presentation, and **Fc receptor**. Modulation of the
expression of MHC class II antigens and other costimulatory molecules,
such as B7 and ICAM-1, may result.

L8 ANSWER 18 OF 61 USPATFULL

AN 2002:81254 USPATFULL

TI Tissue plasminogen activator-like protease

IN Moore, Paul A., Germantown, MD, United States

Ruben, Steven M., Olney, MD, United States

Ebner, Reinhard, Gaithersburg, MD, United States

PA Human Genome Sciences, Inc., Rockville, MD, United States (U.S.
corporation)

PI US 6372473 B1 20020416

AI US 1999-411977 19991004 (9)

RLI Continuation-in-part of Ser. No. US 1998-84491, filed on 27 May 1998

PRAI US 1997-48000P 19970528 (60)

DT Utility

FS GRANTED

EXNAM Primary Examiner: Slobodyansky, Elizabeth

LREP Human Genome Sciences, Inc.

CLMN Number of Claims: 77

ECL Exemplary Claim: 1

DRWN 8 Drawing Figure(s); 8 Drawing Page(s)

LN.CNT 11319

AB The present invention relates to a novel t-PALP protein which is a
member of the serine protease family. In particular, isolated nucleic
acid molecules are provided encoding the human t-PALP protein. t-PALP
polypeptides are also provided as are vectors, host cells and
recombinant methods for producing the same. The invention further

relates to screening methods for identifying agonists and antagonists of t-PALP activity. Also provided are diagnostic methods for detecting circulatory system-related disorders and therapeutic methods for treating circulatory system-related disorders.

DETD . . . Clin. Invest. 79:1440-1446, 1987); anticollagenase-serum; alpha2-antiplasmin (Holmes et al., J. Biol. Chem. 262(4):1659-1664, 1987); Bisantrone (National Cancer Institute); Lobenzarit disodium (N-(2)-**carboxyphenyl**-4-chloroanthronilic acid disodium or "CCA"; Takeuchi et al., Agents Actions 36:312-316, 1992); Thalidomide; Angostatic steroid; AGM-1470; carboxynaminolmidazole; and metalloproteinase inhibitors such. . .

DETD . . . that may be used according to the methods of the invention include, but are not limited to, glutamyl derivatives of **benzoic acid** mustard alkylating agent, phosphate derivatives of etoposide or mitomycin C, cytosine arabinoside, daunorubisin, and phenoxacetamide derivatives of doxorubicin.

DETD . . . Three major families of cell surface antigens can be identified on monocytes: adhesion molecules, molecules involved in antigen presentation, and **Fc receptor**. Modulation of the expression of MHC class II antigens and other costimulatory molecules, such as B7 and ICAM-1, may result. . .

LB ANSWER 19 OF 61 USPATFULL

AN 2002:78442 USPATFULL

TI Nucleic acids, proteins, and antibodies

IN Rosen, Craig A., Laytonsville, MD, UNITED STATES

Ruben, Steven M., Olney, MD, UNITED STATES

Barash, Steven C., Rockville, MD, UNITED STATES

PI US 2002042096 A1 20020411

AI US 2001-764887 A1 20010117 (9)

PRAI US 2000-179065P 20000131 (60)

US 2000-180628P 20000204 (60)

US 2000-214886P 20000628 (60)

US 2000-217487P 20000711 (60)

US 2000-225758P 20000814 (60)

US 2000-220963P 20000726 (60)

US 2000-217496P 20000711 (60)

US 2000-225447P 20000814 (60)

US 2000-218290P 20000714 (60)

US 2000-225757P 20000814 (60)

US 2000-226868P 20000822 (60)

US 2000-216647P 20000707 (60)

US 2000-225267P 20000814 (60)

US 2000-216880P 20000707 (60)

US 2000-225270P 20000814 (60)

US 2000-251869P 20001208 (60)

US 2000-235834P 20000927 (60)

US 2000-234274P 20000921 (60)

US 2000-234223P 20000921 (60)

US 2000-228924P 20000830 (60)

US 2000-224518P 20000814 (60)

US 2000-236369P 20000929 (60)

US 2000-224519P 20000814 (60)

US 2000-220964P 20000726 (60)

US 2000-241809P 20001020 (60)

US 2000-249299P 20001117 (60)

US 2000-236327P 20000929 (60)

US 2000-241785P 20001020 (60)

US 2000-244617P 20001101 (60)

US 2000-225268P 20000814 (60)

US 2000-236368P 20000929 (60)

US 2000-251856P 20001208 (60)

US 2000-251868P 20001208 (60)

US 2000-229344P 20000901 (60)

US 2000-234997P 20000925 (60)

US 2000-229343P 20000901 (60)

US 2000-229345P	20000901 (60)
US 2000-229287P	20000901 (60)
US 2000-229513P	20000905 (60)
US 2000-231413P	20000908 (60)
US 2000-229509P	20000905 (60)
US 2000-236367P	20000929 (60)
US 2000-237039P	20001002 (60)
US 2000-237038P	20001002 (60)
US 2000-236370P	20000929 (60)
US 2000-236802P	20001002 (60)
US 2000-237037P	20001002 (60)
US 2000-237040P	20001002 (60)
US 2000-240960P	20001020 (60)
US 2000-239935P	20001013 (60)

DT Utility

FS APPLICATION

LREP HUMAN GENOME SCIENCES INC, 9410 KEY WEST AVENUE, ROCKVILLE, MD, 20850

CLMN Number of Claims: 24

ECL Exemplary Claim: 1

DRWN No Drawings

LN.CNT 19583

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The present invention relates to novel liver related polynucleotides and the polypeptides encoded by these polynucleotides herein collectively known as "liver antigens," and the use of such liver antigens for detecting disorders of the liver, particularly the presence of cancer of liver and cancer metastases. More specifically, isolated liver associated nucleic acid molecules are provided encoding novel liver associated polypeptides. Novel liver polypeptides and antibodies that bind to these polypeptides are provided. Also provided are vectors, host cells, and recombinant and synthetic methods for producing human liver associated polynucleotides and/or polypeptides. The invention further relates to diagnostic and therapeutic methods useful for diagnosing, treating, preventing and/or prognosing disorders related to the liver, including cancer of liver tissues, and therapeutic methods for treating such disorders. The invention further relates to screening methods for identifying agonists and antagonists of polynucleotides and polypeptides of the invention. The present invention further relates to methods and/or compositions for inhibiting the production and function of the polypeptides of the present invention.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

SUMM . . . that may be used according to the methods of the invention include, but are not limited to, glutamyl derivatives of **benzoic acid** mustard alkylating agent, phosphate derivatives of etoposide or mitomycin C, cytosine arabinoside, daunorubisin, and phenoxacetamide derivatives of doxorubicin.

SUMM . . . Clin. Invest. 79:1440-1446 (1987)); anticollagenase-serum; alpha2-antiplasmin (Holmes et al., J. Biol. Chem. 262(4):1659-1664 (1987)); Bisantrone (National Cancer Institute); Lobenzarit disodium (N-(2)-**carboxyphenyl**-4-chloroanthronilic acid disodium or "CCA"; Takeuchi et al., Agents Actions 36:312-316, 1992); Thalidomide; Angostatic steroid; AGM-1470; carboxynaminolmidazole; and metalloproteinase inhibitors such. . .

SUMM . . . that may be used according to the methods of the invention include, but are not limited to, glutamyl derivatives of **benzoic acid** mustard alkylating agent, phosphate derivatives of etoposide or mitomycin C, cytosine arabinoside, daunorubisin, and phenoxacetamide derivatives of doxorubicin.

DETD . . . Clin. Invest. 79:1440-1446, (1987)); anticollagenase-serum; alpha2-antiplasmin (Holmes et al., J. Biol. Chem. 262(4):1659-1664, (1987)); Bisantrone (National Cancer Institute); Lobenzarit disodium (N-(2)-**carboxyphenyl**-4-chloroanthronilic acid disodium or "CCA"; (Takeuchi et al., Agents Actions 36:312-316, (1992)); and metalloproteinase inhibitors such as BB94.

DETD . . . Three major families of cell surface antigens can be identified on monocytes: adhesion molecules, molecules involved in antigen

presentation, and **Fc receptor**. Modulation of the expression of MHC class II antigens and other costimulatory molecules, such as B7 and ICAM-1, may result. . .

L8 ANSWER 20 OF 61 USPATFULL

AN 2002:72627 USPATFULL

TI Nucleic, acids, proteins, and antibodies

IN Rosen, Craig A., Laytonsville, MD, UNITED STATES

Ruben, Steven M., Olney, MD, UNITED STATES

PI US 2002039764 A1 20020404

AI US 2001-925298 A1 20010810 (9)

RLI Continuation-in-part of Ser. No. WO 2000-US5881, filed on 8 Mar 2000, UNKNOWN

PRAI US 1999-124270P 19990312 (60)

DT Utility

FS APPLICATION

LREP HUMAN GENOME SCIENCES INC, 9410 KEY WEST AVENUE, ROCKVILLE, MD, 20850

CLMN Number of Claims: 23

ECL Exemplary Claim: 1

DRWN No Drawings

LN.CNT 20087

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The present invention relates to novel ovarian cancer and/or breast cancer related polynucleotides, the polypeptides encoded by these polynucleotides herein collectively referred to as "ovarian and/or breast antigens," and antibodies that immunospecifically bind these polypeptides, and the use of such ovarian and/or breast polynucleotides, antigens, and antibodies for detecting, treating, preventing and/or prognosing disorders of the reproductive system, particularly disorders of the ovaries and/or breast, including, but not limited to, the presence of ovarian and/or breast cancer and ovarian and/or breast cancer metastases. More specifically, isolated ovarian and/or breast nucleic acid molecules are provided encoding novel ovarian and/or breast polypeptides. Novel ovarian and/or breast polypeptides and antibodies that bind to these polypeptides are provided. Also provided are vectors, host cells, and recombinant and synthetic methods for producing human ovarian and/or breast polynucleotides, polypeptides, and/or antibodies. The invention further relates to diagnostic and therapeutic methods useful for diagnosing, treating, preventing and/or prognosing disorders related to the ovaries and/or breast, including ovarian and/or breast cancer, and therapeutic methods for treating such disorders. The invention further relates to screening methods for identifying agonists and antagonists of polynucleotides and polypeptides of the invention. The invention further relates to methods and/or compositions for inhibiting or promoting the production and/or function of the polypeptides of the invention.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

SUMM . . . that may be used according to the methods of the invention include, but are not limited to, glutamyl derivatives of **benzoic acid** mustard alkylating agent, phosphate derivatives of etoposide or mitomycin C, cytosine arabinoside, daunorubisin, and phenoxacetamide derivatives of doxorubicin.

SUMM . . . Clin. Invest. 79:1440-1446 (1987)); anticollagenase-serum; alpha2-antiplasmin (Holmes et al., J. Biol. Chem. 262(4):1659-1664 (1987)); Bisantrone (National Cancer Institute); Lobenzarit disodium (N-(2)-**carboxyphenyl**-4-chloroanthronilic acid disodium or "CCA"; Takeuchi et al., Agents Actions 36:312-316, 1992); Thalidomide; Angostatic steroid; AGM-1470; carboxynaminolmidazole; and metalloproteinase inhibitors such. . .

SUMM . . . that may be used according to the methods of the invention include, but are not limited to, glutamyl derivatives of **benzoic acid** mustard alkylating agent, phosphate derivatives of etoposide or mitomycin C, cytosine arabinoside, daunorubisin, and phenoxacetamide derivatives of doxorubicin.

DETD . . . Three major families of cell surface antigens can be identified on monocytes: adhesion molecules, molecules involved in antigen

presentation, and **Fc receptor**. Modulation of the expression of MHC class II antigens and other costimulatory molecules, such as B7 and ICAM-1, may result. . .

L8 ANSWER 21 OF 61 USPATFULL
AN 2002:66904 USPATFULL
TI Fibroblast growth factor-like molecules and uses thereof
IN Jing, Shuqian, Thousand Oaks, CA, UNITED STATES
Bass, Michael Brian, Thousand Oaks, CA, UNITED STATES
PA Amgen, Inc. (U.S. corporation)
PI US 2002037557 A1 20020328
AI US 2001-805805 A1 20010313 (9)
PRAI US 2000-188786P 20000313 (60)
DT Utility
FS APPLICATION
LREP MCDONNELL BOEHNEN HULBERT & BERGHOFF, 300 SOUTH WACKER DRIVE, SUITE
3200, CHICAGO, IL, 60606
CLMN Number of Claims: 54
ECL Exemplary Claim: 1
DRWN 5 Drawing Page(s)
LN.CNT 3796

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The present invention provides Fibroblast Growth Factor-Like (FGF-L) polypeptides and nucleic acid molecules encoding the same. The invention also provides selective binding agents, vectors, host cells, and methods for producing FGF-L polypeptides. The invention further provides pharmaceutical compositions and methods for the diagnosis, treatment, amelioration, and/or prevention of diseases, disorders, and conditions associated with FGF-L polypeptides.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

DETD . . . 337:525-31. When constructed together with a therapeutic protein, an Fc domain can provide longer half-life or incorporate such functions as **Fc receptor** binding, protein A binding, complement fixation, and perhaps even placental transfer. Id. Table II summarizes the use of certain Fc. . .
DETD . . . agents, hydrophilic polymers (such as polyvinylpyrrolidone), low molecular weight polypeptides, salt-forming counterions (such as sodium), preservatives (such as benzalkonium FGF-Loride, **benzoic acid**, salicylic acid, thimerosal, phenethyl alcohol, methylparaben, propylparaben, FGF-Lorhexidine, sorbic acid, or hydrogen peroxide), solvents (such as glycerin, propylene glycol, or. . .

L8 ANSWER 22 OF 61 USPATFULL
AN 2002:66871 USPATFULL
TI IL-17 like molecules and uses thereof
IN Medlock, Eugene, Westlake Village, CA, UNITED STATES
Yeh, Richard, Princeton, NJ, UNITED STATES
Silbiger, Scott M., Woodland Hills, CA, UNITED STATES
Elliott, Gary S., Thousand Oaks, CA, UNITED STATES
Nguyen, Hung Q., Thousand Oaks, CA, UNITED STATES
Jing, Shuqian, Thousand Oaks, CA, UNITED STATES
PI US 2002037524 A1 20020328
AI US 2001-886404 A1 20010621 (9)
RLI Continuation-in-part of Ser. No. US 2001-810384, filed on 16 Mar 2001, PENDING
PRAI US 2001-266159P 20010202 (60)
US 2000-213125P 20000622 (60)
DT Utility
FS APPLICATION
LREP MARSHALL, O'TOOLE, GERSTEIN, MURRAY & BORUN, 6300 SEARS TOWER, 233 SOUTH WACKER DRIVE, CHICAGO, IL, 60606-6402
CLMN Number of Claims: 74
ECL Exemplary Claim: 1
DRWN 26 Drawing Page(s)
LN.CNT 5737

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB Novel IL-17 like polypeptides and nucleic acid molecules encoding the same. The invention also provides vectors, host cells, selective binding agents, and methods for producing IL-17 like polypeptides. Also provided for are methods for the treatment, diagnosis, amelioration, or prevention of diseases with IL-17 like polypeptides, agonists, or antagonists thereof.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

DETD . . . (1989). When constructed together with a therapeutic protein, an Fc domain can provide longer half-life or incorporate such functions as **Fc receptor** binding, protein A binding, complement fixation and perhaps even placental transfer. Id. Table II summarizes the use of certain Fc. . .

DETD . . . agents; hydrophilic polymers (such as polyvinylpyrrolidone); low molecular weight polypeptides; salt-forming counterions (such as sodium); preservatives (such as benzalkonium chloride, **benzoic acid**, salicylic acid, thimerosal, phenethyl alcohol, methylparaben, propylparaben, chlorhexidine, sorbic acid or hydrogen peroxide); solvents (such as glycerin, propylene glycol or. . .

L8 ANSWER 23 OF 61 USPATFULL

AN 2002:57821 USPATFULL

TI Therapeutic inhibitor of vascular smooth muscle cells

IN Kunz, Lawrence L., Redmond, WA, United States

Klein, Richard A., Edmonds, WA, United States

Reno, John M., Brier, WA, United States

PA NeoRx Corporation, Seattle, WA, United States (U.S. corporation).

PI US 6358989 B1 20020319

AI US 1999-361194 19990726 (9)

RLI Division of Ser. No. US 1997-829685, filed on 31 Mar 1997

Continuation-in-part of Ser. No. US 1995-450793, filed on 25 May 1995,

now patented, Pat. No. US 5811447 Continuation of Ser. No. WO

1996-US2125, filed on 15 Feb 1996 Continuation-in-part of Ser. No. US

1995-389712, filed on 15 Feb 1995 Continuation of Ser. No. US

1993-62451, filed on 13 May 1993, now abandoned

DT Utility

FS GRANTED

EXNAM Primary Examiner: Barts, Samuel

LREP Schwegman, Lundenberg, Woessner & Kluth, PA

CLMN Number of Claims: 20

ECL Exemplary Claim: 1

DRWN 30 Drawing Figure(s); 22 Drawing Page(s)

LN.CNT 5403

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB Methods are provided for inhibiting stenosis or restenosis following vascular trauma in a mammalian host, comprising administering to the host a therapeutically effective dosage of a cytostatic agent and/or cytoskeletal inhibitor so as to biologically stent the traumatized vessel. Also provided is a method to inhibit or reduce vascular remodeling following vascular trauma, comprising administering an effective amount of a cytoskeletal inhibitor. Further provided are pharmaceutical compositions and kits comprising the therapeutic agents of the invention.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

DETD . . . include inactive ingredients such as cellulose, pregelatinized starch, silicon dioxide, hydroxypropyl methylcellulose, magnesium stearate, microcrystalline cellulose, starch, talc, titanium dioxide, **benzoic acid**, citric acid, corn starch, mineral oil, polypropylene glycol, sodium phosphate, and zinc stearate, and the like. Hard or soft gelatin. . .

DETD . . . does not bind to sites in the patient through antigen-specific binding, but instead binds in a non-specific manner, e.g., through **Fc receptor** binding reticuloendothelial cells, asialo-receptor binding, and by binding to ubiquitin-expressing cells. The irrelevant "blocker" decreases non-specific binding of the therapeutic. . .

L8 ANSWER 24 OF 61 USPATFULL

AN 2002:51012 USPATFULL

TI **Fc receptor** modulators and uses thereof

IN Baell, Jonathan B., Ivanhoe, AUSTRALIA

Garrett, Thomas P. J., Brunswick, AUSTRALIA

Hogarth, P. Mark, Williamstown, AUSTRALIA

Matthews, Barry R., Olinda, AUSTRALIA

McCarthy, Thomas D., East Malvern, AUSTRALIA

Pietersz, Geoffrey A., Greensborough, AUSTRALIA

PA Ilexus Pty Limited, Victoria, AUSTRALIA (non-U.S. corporation)

PI US 6355683 B1 20020312

AI US 1999-393598 19990910 (9)

PRAI US 1998-99855P 19980911 (60)

US 1999-131938P 19990430 (60)

US 1999-148479P 19990811 (60)

DT Utility

FS GRANTED

EXNAM Primary Examiner: Spivack, Phyllis G.

LREP Sheridan Ross P.C.

CLMN Number of Claims: 50

ECL Exemplary Claim: 1

DRWN 20 Drawing Figure(s); 20 Drawing Page(s)

LN.CNT 1876

AB This invention relates to a pharmaceutical composition comprising a **Fc receptor** modulating compound and a pharmaceutically acceptable carrier. The present invention also relates to a method for treating a variety of diseases using a **Fc receptor** modulating compound.

TI **Fc receptor** modulators and uses thereof

AB This invention relates to a pharmaceutical composition comprising a **Fc receptor** modulating compound and a pharmaceutically acceptable carrier. The present invention also relates to a method for treating a variety of diseases using a **Fc receptor** modulating compound.

DRWD FIGS. 5A-5G show some of the **Fc receptor** modulating compounds including those corresponding to **Fc receptor** modulating activities shown in FIGS. 6-9;

DETD The **Fc receptor** modulating compounds of the present invention can also include nucleosides or derivatives thereof. Preferably, the nucleosides of the present invention.

DETD The **Fc receptor** modulating compounds of the present invention can further include folic acid or its derivatives.

DETD The **Fc receptor** modulating compounds of the present invention can also include peptides which can modulate the interaction between Fc receptors and immunoglobulins..

DETD . . . been found to be effective in modulating the FcR receptor activities. Thus, in another embodiment of the present invention, the **Fc receptor** modulating compound of the present invention also includes a compound of the formula: ##STR26##

DETD In addition to and/or instead of a rational drug design, other **Fc receptor** modulators can be identified by a screening process, where a variety of compounds are tested to determine their **Fc receptor** modulating activity. In this manner, a variety of **Fc receptor** modulators have been identified. Thus, compounds of the present invention include substituted and unsubstituted benzoic acids, in particular, 4-methyl **benzoic acid** and 3-methyl **benzoic acid**; nucleosides and analogs thereof; and folic acid and its derivatives.

DETD The compounds of the present invention are **Fc receptor** modulators, e.g., they modulate **Fc receptor** binding of immunoglobulins. Preferably, the compounds of the present invention modulate Fc receptors selected from the group consisting of Fc.alpha.R.,

DETD This experiment illustrates a synthesis of 1,2-Bis(m-

carboxyphenyl)ethane: ##STR28##

DETD . . . was extracted with EtOAc (3.times.50 mL) and the combined organic extracts dried (Na.sub.2SO.sub.4), filtered and concentrated in vacuo to give 1,2-bis(m-**carboxyphenyl**)ethane as a white solid. MS (APCI) m/z 269 (M+1, 100%). .sup.13C NMR (50 MHz, d.sub.6-DMSO): .delta.38.4, 128.8, 130.3, 131.1, 132.5, . . .

DETD This experiment illustrates a synthesis of 3-[(m-**carboxyphenyl**)methoxy]**benzoic acid: ##STR29##**

DETD Step 2: Using 3-[(m-bromophenyl)methoxy]bromobenzene and the method described in Example 1, step 2 gave 3-[(m-**carboxyphenyl**)methoxy]-**benzoic acid** as a white solid. MS (APCI) m/z 271 (M.sup.+1, 100%). .sup.13C NMR (50 MHz, d.sub.6-DMSO): .delta.68.3, 114.5, 119.3, 121.5, 127.8, . . .

DETD . . . aqueous HCl (1 M, 50 mL). The organic extract was dried (Na.sub.2SO.sub.4), filtered and concentrated in vacuo to give 1,3-bis(m-**carboxyphenyl**)-1-propanol as a viscous oil. MS (APCI) m/z 299 (M.sup.+1, 100%). .sup.1H NMR (200 MHz, CDCl.sub.3); .delta.1.95-2.10, m, 2H; 2.68-2.83, m, . . .

DETD This experiment illustrates a synthesis of (S,S)-1,2-bis-(3-**carboxyphenyl**)ethane-1,2-diol: **##STR34##**

DETD . . . The above diester (500 mg, 1.5 mmol) was hydrolyzed using the procedure described in Example 6, step 2 to give (S,S)-1,2-bis-(3-**carboxyphenyl**)ethane-1,2-diol as a white solid. MS (APCI) m/z 301 (M.sup.+1, 100%). .sup.1H NMR (200 MHz, d.sub.6-DMSO): .delta.3.40, bs, 1H; 4.76, s, . . .

DETD Step 3: The ester in Step 2 was hydrolyzed using the procedure described in Example 6, step 2 to give 1-[m-(carboxymethyl)phenyl]-2-[m-(**carboxyphenyl**)]ethane as a white solid. MS (APCI) m/z 283 (M.sup.+1, 100%). .sup.1H NMR (200 MHz, d.sub.6-DMSO): .delta.2.92, m, 4H; 3.55, s, . . .

DETD This experiment illustrates **Fc receptor** modulating activity of some of the compounds of the present invention.

DETD This experiment illustrates a synthesis of N-(3'-**carboxyphenyl**)-2-(carboxybenzene)sulfonamide: **##STR41##**

DETD . . . The above diester (1.0 g, 2.75 mmol) was hydrolyzed using the procedure described in Example 6, step 2 to provide N-(3'-**carboxyphenyl**)-2-(carboxybenzene)sulfonamide as a white solid. MS (CI) m/z 320 (M.sup.+1, 100%). .sup.13C NMR (50 MHz, d.sub.6-DMSO): .delta.168.0, 166.3, 137.3, 135.8, 133.4, . . .

DETD This experiment illustrates a synthesis of (3R,4R)-4,5-bis(m-**carboxyphenyl**)imidazolid-2-one: **##STR44##**

DETD . . . The above diester (68 mg, 0.19 mmol) was hydrolyzed using the procedure described in Example 6, step 2 to give (3R,4R)-4,5-bis(m-**carboxyphenyl**)imidazolid-2-one as a white solid. MS (electrospray) m/z 327 (M.sup.++1, 100%). .sup.13C NMR (50 MHz, d.sub.6-DMSO): .delta.64.3, 127.4, 129.1, 129.3, 131.1, . . .

DETD This experiment illustrates **Fc receptor** modulating activity of a tripeptide and a hexapeptide.

DETD For each aggregation experiment, a mixture of 50 .mu.L of the **Fc receptor** agonist, heat aggregated gamma globulin ("HAGG", 200 .mu.g/mL) or collagen (2 .mu.g/mL) was incubated with 50 .mu.L of phosphate buffered. . .

CLM What is claimed is:

24. A method for inhibiting **Fc receptor** binding of immunoglobulin in a patient comprising administering to such patient a pharmaceutically effective amount of a compound of the . . .

25. The method of claim 24, wherein said **Fc receptor** is selected from the group consisting of Fc.alpha.R, Fc.epsilon.R, Fc.gamma.R and mixtures thereof.

26. The method of claim 25, wherein said **Fc receptor** is selected from the group consisting of Fc.gamma.RIIa, Fc.gamma.RIIb, Fc.gamma.RIIc and mixtures thereof.

TI 49 human secreted proteins
IN Moore, Paul A., Germantown, MD, UNITED STATES
Ruben, Steven M., Olney, MD, UNITED STATES
Olsen, Henrik S., Gaithersburg, MD, UNITED STATES
Shi, Yanggu, Gaithersburg, MD, UNITED STATES
Rosen, Craig A., Laytonsville, MD, UNITED STATES
Florence, Kimberly A., Rockville, MD, UNITED STATES
Soppet, Daniel R., Centreville, VA, UNITED STATES
LaFleur, David W., Washington, DC, UNITED STATES
Endress, Gregory A., Potomac, MD, UNITED STATES
Ebner, Reinhard, Gaithersburg, MD, UNITED STATES
Komatsoulis, George, Silver Spring, MD, UNITED STATES
Duan, Roxanne D., Bethesda, MD, UNITED STATES
PI US 2002026040 A1 20020228
AI US 2001-904615 A1 20010716 (9)
RLI Continuation of Ser. No. US 2000-739254, filed on 19 Dec 2000, PENDING
Continuation of Ser. No. US 2000-511554, filed on 23 Feb 2000, ABANDONED
Continuation-in-part of Ser. No. WO 1999-US19330, filed on 24 Aug 1999,
UNKNOWN
PRAI US 1998-97917P 19980825 (60)
US 1998-98634P 19980831 (60)
DT Utility
FS APPLICATION
LREP HUMAN GENOME SCIENCES INC, 9410 KEY WEST AVENUE, ROCKVILLE, MD, 20850
CLMN Number of Claims: 23
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 19401

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The present invention relates to novel human secreted proteins and isolated nucleic acids containing the coding regions of the genes encoding such proteins. Also provided are vectors, host cells, antibodies, and recombinant methods for producing human secreted proteins. The invention further relates to diagnostic and therapeutic methods useful for diagnosing and treating diseases, disorders, and/or conditions related to these novel human secreted proteins.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

SUMM . . . Clin. Invest. 79:1440-1446, 1987); anticollagenase-serum; alpha2-antiplasmin (Holmes et al., J. Biol. Chem. 262(4):1659-1664, 1987); Bisantrene (National Cancer Institute); Lobenzarit disodium (N-(2)-**carboxyphenyl**-4-chloroanthronilic acid disodium or "CCA"; Takeuchi et al., Agents Actions 36:312-316, 1992); Thalidomide; Angostatic steroid; AGM-1470; carboxynaminolmidazole; and metalloproteinase inhibitors such. . .

SUMM . . . that may be used according to the methods of the invention include, but are not limited to, glutamyl derivatives of **benzoic acid** mustard alkylating agent, phosphate derivatives of etoposide or mitomycin C, cytosine arabinoside, daunorubisin, and phenoxyacetamide derivatives of doxorubicin.

DETD . . . Three major families of cell surface antigens can be identified on monocytes: adhesion molecules, molecules involved in antigen presentation, and **Fc receptor**. Modulation of the expression of MHC class II antigens and other costimulatory molecules, such as B7 and ICAM-1, may result. . .

L8 ANSWER 26 OF 61 USPATFULL

AN 2002:43612 USPATFULL

TI Therapeutic inhibitor of vascular smooth muscle cells

IN Kunz, Lawrence L., Redmond, WA, UNITED STATES

Reno, John M., Brier, WA, UNITED STATES

PI US 2002025979 A1 20020228

AI US 2001-896208 A1 20010629 (9)

RLI Division of Ser. No. US 1997-829991, filed on 31 Mar 1997, PENDING
Continuation-in-part of Ser. No. US 1995-450793, filed on 25 May 1995,
GRANTED, Pat. No. US 5811447 Continuation of Ser. No. US 1993-62451,
filed on 13 May 1993, ABANDONED Continuation of Ser. No. WO 1996-US2125,

filed on 15 Feb 1996, UNKNOWN Continuation-in-part of Ser. No. US
1995-389712, filed on 15 Feb 1995, PENDING

DT Utility
FS APPLICATION
LREP SCHWEGMAN, LUNDBERG, WOESSNER & KLUTH, P.A., 1600 TCF TOWER, 121 SOUTH
8TH STREET, MINNEAPOLIS, MN, 55402
CLMN Number of Claims: 60
ECL Exemplary Claim: 1
DRWN 22 Drawing Page(s)
LN.CNT 5068

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB Methods are provided for inhibiting stenosis or restenosis following
vascular trauma in a mammalian host, comprising administering to the
host a therapeutically effective dosage of a cytostatic agent and/or
cytoskeletal inhibitor so as to biologically stent the traumatized
vessel. Also provided is a method to inhibit or reduce vascular
remodeling following vascular trauma, comprising administering an
effective amount of a cytoskeletal inhibitor. Further provided are
pharmaceutical compositions and kits comprising the therapeutic agents
of the invention.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

DETD . . . include inactive ingredients such as cellulose, pregelatinized
starch, silicon dioxide, hydroxypropyl methylcellulose, magnesium
stearate, microcrystalline cellulose, starch, talc, titanium dioxide,
benzoic acid, citric acid, corn starch, mineral oil,
polypropylene glycol, sodium phosphate, and zinc stearate, and the like.
Hard or soft gelatin. . .

DETD . . . does not bind to sites in the patient through antigen-specific
binding, but instead binds in a non-specific manner, e.g., through
Fc receptor binding reticuloendothelial cells,
asialo-receptor binding, and by binding to ubiquitin-expressing cells.
The irrelevant "blocker" decreases non-specific binding of the
therapeutic. . .

L8 ANSWER 27 OF 61 USPATFULL

AN 2002:27135 USPATFULL

TI Beta-like glycoprotein hormone polypeptide and heterodimer

IN Paszty, Christopher J.R., Ventura, CA, UNITED STATES

Cao, Jin, Tarzana, CA, UNITED STATES

Danilenko, Dimitry M., Thousand Oaks, CA, UNITED STATES

Gong, Jianhua, Thousand Oaks, CA, UNITED STATES

Hill, David C., Thousand Oaks, CA, UNITED STATES

PI US 2002015981 A1 20020207

AI US 2001-818954 A1 20010327 (9)

RLI Continuation-in-part of Ser. No. US 2000-723970, filed on 27 Nov 2000,
PENDING

PRAI US 2000-199211P 20000424 (60)

US 2000-192654P 20000328 (60)

DT Utility

FS APPLICATION

LREP AMGEN INCORPORATED, MAIL STOP 27-4-A, ONE AMGEN CENTER DRIVE, THOUSAND
OAKS, CA, 91320-1799

CLMN Number of Claims: 99

ECL Exemplary Claim: 1

DRWN 7 Drawing Page(s)

LN.CNT 4778

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB Novel .beta.10 polypeptides and heterodimers thereof, and nucleic acid
molecules encoding the same are disclosed. The invention also provides
vectors, host cells, selective binding agents, and methods for producing
.beta.10 polypeptides and heterodimeric forms thereof, specifically
.alpha.2/.beta.10. Also provided for are methods for the treatment,
diagnosis, amelioration, or prevention of diseases with .beta.10
polypeptides and .alpha.2/.beta.10 heterodimers or their respective
binding agents.

This application is a continuation-in-part of U.S. application Ser. No. 09/723,970, filed Nov. 27, 2000, which claims the benefit of U.S. Provisional Application Ser. No. 60/199,211, filed Apr. 24, 2000, and U.S. Provisional Application Ser. No. 60/192,654, filed Mar. 28, 2000, which are hereby incorporated by reference.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

DETD . . . (1989). When constructed together with a therapeutic protein, an Fc domain can provide longer half-life or incorporate such functions as **Fc receptor** binding, protein A binding, complement fixation and perhaps even placental transfer. Id. Table II summarizes the use of certain Fc. . . .

DETD . . . agents, hydrophilic polymers (such as polyvinylpyrrolidone), low molecular weight polypeptides, salt-forming counterions (such as sodium), preservatives (such as benzalkonium chloride, **benzoic acid**, salicylic acid, thimerosal, phenethyl alcohol, methylparaben, propylparaben, chlorhexidine, sorbic acid or hydrogen peroxide), solvents (such as glycerin, propylene glycol or. . . .

L8 ANSWER 28 OF 61 USPATFULL
AN 2002:22131 USPATFULL
TI 18 Human secreted proteins
IN Shi, Yanggu, Gaithersburg, MD, UNITED STATES
Young, Paul E., Gaithersburg, MD, UNITED STATES
Ebner, Reinhard, Gaithersburg, MD, UNITED STATES
Soppet, Daniel R., Centreville, VA, UNITED STATES
Ruben, Steven M., Olney, MD, UNITED STATES
PI US 2002012966 A1 20020131
AI US 2001-768826 A1 20010125 (9)
RLI Continuation-in-part of Ser. No. WO 2000-US22350, filed on 15 Aug 2000, UNKNOWN
PRAI US 1999-148759P 19990816 (60)
DT Utility
FS APPLICATION
LREP HUMAN GENOME SCIENCES INC, 9410 KEY WEST AVENUE, ROCKVILLE, MD, 20850
CLMN Number of Claims: 23
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 18157

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The present invention relates to novel human secreted proteins and isolated nucleic acids containing the coding regions of the genes encoding such proteins. Also provided are vectors, host cells, antibodies, and recombinant methods for producing human secreted proteins. The invention further relates to diagnostic and therapeutic methods useful for diagnosing and treating diseases, disorders, and/or conditions related to these novel human secreted proteins.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

SUMM . . . Clin. Invest. 79:1440-1446, 1987); anticollagenase-serum; alpha2-antiplasmin (Holmes et al., J. Biol. Chem. 262(4):1659-1664, 1987); Bisantrene (National Cancer Institute); Lobenzarit disodium (N-(2)-**carboxyphenyl**-4-chloroanthronilic acid disodium or "CCA"; Takeuchi et al., Agents Actions 36:312-316, 1992); Thalidomide; Angostatic steroid; AGM-1470; carboxynaminolmidazole; and metalloproteinase inhibitors such. . . .

SUMM . . . that may be used according to the methods of the invention include, but are not limited to, glutamyl derivatives of **benzoic acid** mustard alkylating agent, phosphate derivatives of etoposide or mitomycin C, cytosine arabinoside, daunorubisin, and phenoxyacetamide derivatives of doxorubicin.

DETD . . . Clin. Invest. 79:1440-1446, (1987)); anticollagenase-serum; alpha2-antiplasmin (Holmes et al., J. Biol. Chem. 262(4):1659-1664, (1987)); Bisantrene (National Cancer Institute); Lobenzarit disodium (N-(2)-**carboxyphenyl**-4-chloroanthronilic acid disodium or "CCA"; (Takeuchi et al., Agents Actions 36:312-316, (1992)); and metalloproteinase inhibitors such as BB94.

DETD . . . Three major families of cell surface antigens can be identified on monocytes: adhesion molecules, molecules involved in antigen presentation, and **Fc receptor**. Modulation of the expression of MHC class II antigens and other costimulatory molecules, such as B7 and ICAM-1, may result. . .

L8 ANSWER 29 OF 61 USPATFULL

AN 2002:19196 USPATFULL

TI Eukaryotic layered vector initiation systems for production of recombinant proteins

IN Dubensky, Jr., Thomas W., Rancho Sante Fe, CA, United States

Polo, John M., San Diego, CA, United States

Driver, David A., San Diego, CA, United States

PA Chiron Corporation, Emeryville, CA, United States (U.S. corporation)

PI US 6342372 B1 20020129

AI US 1999-350399 19990708 (9)

RLI Continuation of Ser. No. US 1997-931783, filed on 16 Sep 1997, now abandoned Division of Ser. No. US 1995-404796, filed on 15 Mar 1995, now patented, Pat. No. US 6015686 Continuation-in-part of Ser. No. US 1995-376184, filed on 20 Jan 1995, now abandoned Continuation-in-part of Ser. No. US 1994-348472, filed on 30 Nov 1994, now abandoned Continuation-in-part of Ser. No. US 1994-198450, filed on 18 Feb 1994, now abandoned Continuation-in-part of Ser. No. US 1993-122791, filed on 15 Sep 1993, now abandoned

DT Utility

FS GRANTED

EXNAM Primary Examiner: Brusca, John S.

LREP McMasters, David D., Dollard, Anne S., Blackburn, Robert P.

CLMN Number of Claims: 14

ECL Exemplary Claim: 1

DRWN 37 Drawing Figure(s); 30 Drawing Page(s)

LN.CNT 10217

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The present invention provides compositions and methods for utilizing recombinant alphavirus vectors. Also disclosed are compositions and methods for making and utilizing eukaryotic layered vector initiation systems.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

DETD . . . 89:33, 1992); carboxypeptidase G2, which will cleave the glutamic acid from para-N-bis (2-chloroethyl) aminobenzoyl glutamic acid, thereby creating a toxic **benzoic acid** mustard; and Penicillin-V amidase, which will convert phenoxycetabide derivatives of doxorubicin and melphalan to toxic compounds (see generally, Vrudhula et. . .

DETD . . . proteins that recognize Fc portions of antibodies. Monoclonal antibodies which recognize only preselected target cells are then bound to such **Fc receptor**-bearing alphavirus vector particles, such that the vector particles bind to and infect only those preselected target cells (for example, tumor. . .

L8 ANSWER 30 OF 61 USPATFULL

AN 2002:16896 USPATFULL

TI Fibroblast growth factor receptor-like molecules and uses thereof

IN Saris, Christiaan M., Newbury Park, CA, UNITED STATES

Mu, Sharon X., Thousand Oaks, CA, UNITED STATES

Xia, Min, Newbury Park, CA, UNITED STATES

Boone, Thomas Charles, Newbury Park, CA, UNITED STATES

Covey, Todd, Moorpark, CA, UNITED STATES

PA Amgen, Inc. (U.S. corporation)

PI US 2002009776 A1 20020124

AI US 2001-815108 A1 20010322 (9)

PRAI US 2000-191379P 20000322 (60)

DT Utility

FS APPLICATION

LREP MCDONNELL BOEHNEN HULBERT & BERGHOFF, 300 SOUTH WACKER DRIVE, SUITE 3200, CHICAGO, IL, 60606

CLMN Number of Claims: 56
ECL Exemplary Claim: 1
DRWN 30 Drawing Page(s)
LN.CNT 4217

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The present invention provides Fibroblast Growth Factor Receptor-Like (FGFR-L) polypeptides and nucleic acid molecules encoding the same. The invention also provides selective binding agents, vectors, host cells, and methods for producing FGFR-L polypeptides. The invention further provides pharmaceutical compositions and methods for the diagnosis, treatment, amelioration, and/or prevention of diseases, disorders, and conditions associated with FGFR-L polypeptides.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

DETD . . . 337:525-31. When constructed together with a therapeutic protein, an Fc domain can provide longer half-life or incorporate such functions as **Fc receptor** binding, protein A binding, complement fixation, and perhaps even placental transfer. Id. Table II summarizes the use of certain Fc: . . .

DETD . . . agents, hydrophilic polymers (such as polyvinylpyrrolidone), low molecular weight polypeptides, salt-forming counterions (such as sodium), preservatives (such as benzalkonium FGFR-Loride, **benzoic acid**, salicylic acid, thimerosal, phenethyl alcohol, methylparaben, propylparaben, FGFR-Lorhexidine, sorbic acid, or hydrogen peroxide), solvents (such as glycerin, propylene glycol, or. . .

L8 ANSWER 31 OF 61 USPATFULL

AN 2002:16578 USPATFULL

TI Composition and method for treating inflammatory diseases

IN Boone, Thomas C., Newbury Park, CA, UNITED STATES

Hershenson, Susan, Newbury Park, CA, UNITED STATES

Bevilacqua, Michael P., Boulder, CO, UNITED STATES

Collins, David S., Fishers, IN, UNITED STATES

PA Amgen Inc. (U.S. corporation)

PI US 2002009454 A1 20020124

AI US 2001-784623 A1 20010215 (9)

RLI Division of Ser. No. US 1998-131247, filed on 7 Aug 1998, PENDING

PRAI WO 1997-US2131 19970210

US 1997-55185P 19970808 (60)

DT Utility

FS APPLICATION

LREP Timothy J. Gaul, U.S. Patent Operations/TJG, Dept. 4300, M/S 27-4-A, AMGEN, INC., One Amgen Center Drive, Thousand Oaks, CA, 91320-1799

CLMN Number of Claims: 20

ECL Exemplary Claim: 1

DRWN 14 Drawing Page(s)

LN.CNT 3525

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB A protein which exhibits a therapeutic effect on inflammation and is useful for treating IL-1-mediated inflammatory diseases, particularly diseases of the joint.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

SUMM . . . Therapeutic protein products have been constructed using the Fc domain to provide longer half-life or to incorporate functions such as **Fc receptor** binding, protein A binding, complement fixation and placental transfer which all reside in the Fc proteins of immunoglobulins. Id. For. . .

DETD [0044] Modifications may be made to introduce four amino acid substitutions to ablate the **Fc receptor** binding site and the complement (Clq) binding site.

DETD . . . and pluronics); viscosity; clarity; color; sterility; stability (e.g., sucrose and sorbitol); antioxidants (e.g., sodium sulfite and sodium hydrogen-sulfite); preservatives (e.g., **benzoic acid** and salicylic acid); odor of the formulation; flavoring and diluting agents; rate of dissolution (e.g., solubilizers or solubilizing

agents such. . .

L8 ANSWER 32 OF 61 USPATFULL
AN 2002:9650 USPATFULL
TI Method of suppressing an immune response to a transplanted organ or tissue by administering an OX-2 protein
IN Gorczynski, Reginald M., Willowdale, CANADA
PA Transplantation Technologies Inc., Toronto, CANADA (non-U.S. corporation)
PI US 6338851 B1 20020115
AI US 2000-570367 20000505 (9)
RLI Continuation of Ser. No. WO 1998-CA1038, filed on 6 Nov 1998
PRAI US 1997-64764P 19971107 (60)
DT Utility
FS GRANTED
EXNAM Primary Examiner: Gambel, Phillip; Assistant Examiner: Roark, Jessica H.
LREP Bereskin & Parr, Gravelle, Micheline
CLMN Number of Claims: 14
ECL Exemplary Claim: 1
DRWN 20 Drawing Figure(s); 18 Drawing Page(s)
LN.CNT 3129

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB Methods and compositions for inducing immune suppression are disclosed. The methods involve administering an effective amount of an OX-2 protein or a nucleic acid encoding an OX-2 protein. The methods are useful in preventing graft rejection, fetal loss, autoimmune disease, and allergies. Methods and compositions for preventing immune suppression are also disclosed. The methods involve administering an effective amount of an agent that inhibits OX-2..

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

DETD . . . acetic acid, propionic acid, glycolic acid, lactic acid, pyruvic acid, oxalic acid, succinic acid, malic acid, tartaric acid, citric acid, **benzoic acid**, salicylic acid, benzenesulphonic acid, and tolunesulphonic acids.
DETD . . . seen with a murine IgG1 isotype control (BALB/c anti-TNP, clone 107.3: unpublished), making it unlikely that the band observed was **Fc receptor**.

L8 ANSWER 33 OF 61 USPATFULL
AN 2002:3850 USPATFULL
TI Fibroblast growth factor-like molecules and uses thereof
IN Itoh, Nobuyuki, Otsu, JAPAN
PI US 2002001825 A1 20020103
AI US 2001-822485 A1 20010402 (9)
RLI Continuation-in-part of Ser. No. US 2000-540118, filed on 31 Mar 2000, PENDING
DT Utility
FS APPLICATION
LREP FINNEGAN, HENDERSON, FARABOW,, GARRETT & DUNNER, L.L.P., 1300 I Street, N.W., Washington, DC, 20005
CLMN Number of Claims: 45
ECL Exemplary Claim: 1
DRWN 6 Drawing Page(s)
LN.CNT 4409

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB Novel FGF-like polypeptides and nucleic acid molecules encoding the same. The invention also provides vectors, host cells, selective binding agents, and methods for producing FGF-like polypeptides. Also provided for are methods for the treatment, diagnosis, amelioration, or prevention of diseases with FGF-like polypeptides.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

DETD . . . (1989). When constructed together with a therapeutic protein, an Fc domain can provide longer half-life or incorporate such functions as **Fc receptor** binding, protein A binding, complement fixation and perhaps even placental transfer. Id. Table II

summarizes the use of certain Fc. . .
DETD . . . agents, hydrophilic polymers (such as polyvinylpyrrolidone),
low molecular weight polypeptides, salt-forming counterions (such as
sodium), preservatives (such as benzalkonium chloride, **benzoic**
acid, salicylic acid, thimerosal, phenethyl alcohol,
methylparaben, propylparaben, chlorhexidine, sorbic acid or hydrogen
peroxide), solvents (such as glycerin, propylene glycol or. . .

L8 ANSWER 34 OF 61 USPATFULL

AN 2001:231292 USPATFULL

TI Substituted imidazolidine derivatives, their preparation, their use and
pharmaceutical preparations including them

IN Wehner, Volkmar, Sandberg, Germany, Federal Republic of
Stilz, Hans Ulrich, Frankfurt, Germany, Federal Republic of
Schmidt, Wolfgang, Frankfurt, Germany, Federal Republic of
Seiffge, Dirk, Mainz-Kostheim, Germany, Federal Republic of

PA Aventis Pharma Deutschland GmbH, Frankfurt am Main, Germany, Federal
Republic of (non-U.S. corporation)

PI US 6331552 B1 20011218

AI US 2000-516587 20000301 (9)

RLI Continuation of Ser. No. US 1998-195440, filed on 18 Nov 1998, now
abandoned

PRAI DE 1997-19751251 19971119

DT Utility

FS GRANTED

EXNAM Primary Examiner: Oswecki, Jane C.

LREP Heller Ehrman White & McAuliffe LLP

CLMN Number of Claims: 33

ECL Exemplary Claim: 1

DRWN No Drawings

LN.CNT 5731

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB Substituted imidazolidine derivatives of the formula I, ##STR1##

in which B, E, W, Y, R, R.sup.2, R.sup.3, R.sup.30, e and h have the
meanings indicated in the claims. The compounds of the formula I are
valuable pharmaceutical active compounds, which are suitable, for
example, for the therapy of inflammatory disorders, for example of
rheumatoid arthritis, or of allergic disorders. The compounds of the
formula I are inhibitors of the adhesion and migration of leucocytes
and/or antagonists of the adhesion receptor VLA-4 belonging to the
integrins group. They are generally suitable for the therapy or
prophylaxis of illnesses which are caused by an undesired extent of
leucocyte adhesion and/or leucocyte migration or are associated
therewith, or in which cell-cell or cell-matrix interactions which are
based on interactions of VLA-4 receptors with their ligands play a part.
The invention furthermore relates to processes for the preparation of
the compounds of the formula I, their use, in particular as
pharmaceutical active compounds, and pharmaceutical preparations which
contain compounds of the formula I.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

SUMM . . . acid or phosphoric acid, and with organic carboxylic acids or
sulfonic acids, such as, for example, acetic acid, citric acid,
benzoic acid, maleic acid, fumaric acid, tartaric
acid, methanesulfonic acid or p-toluenesulfonic acid. Compounds which
contain both acidic groups and basic groups. . .

DETD 2.4 The plates were incubated at room temperature for 20 minutes with
100 .mu.l/well of **Fc receptor** blocking buffer (1
mg/ml of .gamma.-globulin, 100 mM NaCl, 100 .mu.M MgCl.sub.2, 100 .mu.M
MnCl.sub.2, 100 .mu.M CaCl.sub.2, 1 mg/ml of BSA in 50 mM HEPES, pH
7.5). After removal of the **Fc receptor** blocking
buffer, washing was carried out once with PBS.

DETD 2.6 U937 cells were incubated in **Fc receptor**
blocking buffer for 20 minutes and then added by pipette in a
concentration of 1.times.10.sup.6 /ml and in an amount. . .

L8 ANSWER 35 OF 61 USPATFULL
AN 2001:184866 USPATFULL
TI Therapeutic inhibitor of vascular smooth muscle cells
IN Kunz, Lawrence L., Redmond, WA, United States
Reno, John M., Brier, WA, United States
PA NeoRx Corporation, Seattle, WA, United States (U.S. corporation)
PI US 6306421 B1 20011023
AI US 1997-829991 19970331 (8)
RLI Continuation-in-part of Ser. No. US 1995-450793, filed on 25 May 1995, now patented, Pat. No. US 5811447 Continuation of Ser. No. US 1993-62451, filed on 13 May 1993, now abandoned Continuation-in-part of Ser. No. US 1993-11669, filed on 28 Jan 1993 Continuation-in-part of Ser. No. WO 1992-US8220, filed on 25 Sep 1992 Continuation-in-part of Ser. No. WO 1996-US2125, filed on 15 Feb 1996 Continuation-in-part of Ser. No. US 1995-389712, filed on 15 Feb 1995, now abandoned
DT Utility
FS GRANTED
EXNAM Primary Examiner: Barts, Samuel
LREP Schwegman, Lundberg, Woessner & Kluth, P.A.
CLMN Number of Claims: 36
ECL Exemplary Claim: 1
DRWN 30 Drawing Figure(s); 22 Drawing Page(s)
LN.CNT 5649

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB Methods are provided for inhibiting stenosis or restenosis following vascular trauma in a mammalian host, comprising administering to the host a therapeutically effective dosage of a cytostatic agent and/or cytoskeletal inhibitor so as to biologically stent the traumatized vessel. Also provided is a method to inhibit or reduce vascular remodeling following vascular trauma, comprising administering an effective amount of a cytoskeletal inhibitor. Further provided are pharmaceutical compositions and kits comprising the therapeutic agents of the invention.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

DETD . . . include inactive ingredients such as cellulose, pregelatinized starch, silicon dioxide, hydroxypropyl methylcellulose, magnesium stearate, microcrystalline cellulose, starch, talc, titanium dioxide, **benzoic acid**, citric acid, corn starch, mineral oil, polypropylene glycol, sodium phosphate, and zinc stearate, and the like. Hard or soft gelatin. . .

DETD . . . does not bind to sites in the patient through antigen-specific binding, but instead binds in a non-specific manner, e.g., through **Fc receptor** binding reticuloendothelial cells, asialo-receptor binding, and by binding to ubiquitin-expressing cells. The irrelevant "blocker" decreases non-specific binding of the therapeutic. . .

L8 ANSWER 36 OF 61 USPATFULL
AN 2001:165439 USPATFULL
TI Method to enhance the immunogenicity of an antigen
IN Cowing, Carol O., Del Mar, CA, United States
PI US 2001024649 A1 20010927
AI US 2001-809158 A1 20010315 (9)
RLI Continuation-in-part of Ser. No. US 1998-176044, filed on 20 Oct 1998, GRANTED, Pat. No. US 6210672
DT Utility
FS APPLICATION
LREP KNOBBE MARTENS OLSON & BEAR LLP, 620 NEWPORT CENTER DRIVE, SIXTEENTH FLOOR, NEWPORT BEACH, CA, 92660
CLMN Number of Claims: 57
ECL Exemplary Claim: 1
DRWN 14 Drawing Page(s)
LN.CNT 1919

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The present invention is related to a method for enhancing the immunogenicity of an antigen in a mammal by introducing into the mammal

an antigen or a portion thereof and administering to the mammal a treatment that increases antigen presentation in a lymphoid organ.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

SUMM . . . the lipophilic molecule may be selected from the group consisting of dibutyl phthalate, dibutyl-D-tartarate, N,N-diethyl-toluamide, dibutylfumarate, di(2-ethylhexyl)fumarate, diisooctylmaleate, diethylhexylmaleate, diisooctylfumarate, **benzoic acid**, bihenylmaleate, dioctylphthalate, dibutylmaleate, dioctymaleate, dibutylsuccinate, dioctylsuccinate, dinonylphthalate, diisononylphthalate, dimethylphthalate, diethylphthalate, dipropylphthalate, diphenylphthalate, dibenzylbutylphthalate, diethylmethylphthalate and camphor.

DETD . . . Abbreviation Compound

DBP	dibutyl phthalate
DBT	dibutyl-D-tartarate
DET	N,N-diethyl-toluamide
DBF	dibutylfumarate
DEHF	di(2-ethylhexyl)fumarate
DIOM	diisooctylmaleate
DEHM	di(ethylhexyl)maleate
DIOF	disooctylfumarate
BA	benzoic acid
C	camphor
BM	bihenylmaleate
DOP	dioctylphthalate
DBM	dibutylmaleate
DOM	dioctylmaleate
DBS	dibutylsuccinate
DOS	dioctylsuccinate
DNP	dinonylphthalate
DINP	diisononylphthalate
DMP	dimethylphthalate

DETD . . . mixture of isotype control mAbs (rat IgG2a, rat IgG2b, mouse IgG1 and mouse IgG2a, each at 100-200 .mu.g/ml) and the anti-**Fc receptor** mAb 2.4G2. Langerhans cells were identified by their characteristic light scatter properties (high forward, moderate side scatter) and exceptionally high. . . .

CLM What is claimed is:

. . . wherein the lipophilic molecule is selected from the group consisting of dibutyl phthalate, dibutyl-D-tartarate, N,N-diethyl-toluamide, dibutylfumarate, di(2-ethylhexyl)fumarate, diisooctylmaleate, diethylhexylmaleate, diisooctylfumarate, **benzoic acid**, bihenylmaleate, dioctylphthalate, dibutylmaleate, dioctymaleate, dibutylsuccinate, dioctylsuccinate, dinonylphthalate, diisononylphthalate, dimethylphthalate, diethylphthalate, dipropylphthalate, diphenylphthalate, dibenzylbutylphthalate, diethylmethylphthalate and camphor.

. . . wherein the lipophilic molecule is selected from the group consisting of dibutyl phthalate, dibutyl-D-tartarate, N,N-diethyl-toluamide, dibutylfumarate, di(2-ethylhexyl)fumarate, diisooctylmaleate, diethylhexylmaleate, diisooctylfumarate, **benzoic acid**, bihenylmaleate, dioctylphthalate, dibutylmaleate, dioctymaleate, dibutylsuccinate, dioctylsuccinate, dinonylphthalate, diisononylphthalate, dimethylphthalate, diethylphthalate, dipropylphthalate, diphenylphthalate, dibenzylbutylphthalate, diethylmethylphthalate and camphor.

L8 ANSWER 37 OF 61 USPATFULL

AN 2001:162845 USPATFULL

TI Composition and method for treating inflammatory diseases

IN Boone, Thomas C., Newbury Park, CA, United States

Hershenson, Susan, Newbury Park, CA, United States

Bevilacqua, Michael P., Boulder, CO, United States
Collins, David S., Fishers, IN, United States
PA Amgen Inc., Thousand Oaks, CA, United States (U.S. corporation)
PI US 6294170 B1 20010925
AI US 1998-131247 19980807 (9)
PRAI US 1997-55185P 19970808 (60)
DT Utility
FS GRANTED
EXNAM Primary Examiner: Born, Michael
LREP Gaul, Timothy J., Levy, Ron K., Odre, Steven M.
CLMN Number of Claims: 15
ECL Exemplary Claim: 1
DRWN 14 Drawing Figure(s); 14 Drawing Page(s)
LN.CNT 3022

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB A protein which exhibits a therapeutic effect on inflammation and is useful for treating IL-1-mediated inflammatory diseases, particularly diseases of the joint.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

SUMM Therapeutic protein products have been constructed using the Fc domain to provide longer half-life or to incorporate functions such as **Fc receptor** binding, protein A binding, complement fixation and placental transfer which all reside in the Fc proteins of immunoglobulins. Id. For. . . .

DETD Modifications may be made to introduce four amino acid substitutions to ablate the **Fc receptor** binding site and the complement (C1q) binding site.

DETD . . . and pluronics); viscosity; clarity; color; sterility; stability (e.g., sucrose and sorbitol); antioxidants (e.g., sodium sulfite and sodium hydrogen-sulfite); preservatives (e.g., **benzoic acid** and salicylic acid); odor of the formulation; flavoring and diluting agents; rate of dissolution (e.g., solubilizers or solubilizing agents such. . . .

L8 ANSWER 38 OF 61 USPATFULL

AN 2001:155766 USPATFULL

TI 49 human secreted proteins

IN Moore, Paul A., Germantown, MD, United States
Ruben, Steven M., Oley, MD, United States
Olsen, Henrik S., Gaithersburg, MD, United States
Shi, Yanggu, Gaithersburg, MD, United States
Rosen, Craig A., Laytonsville, MD, United States
Florence, Kimberly A., Rockville, MD, United States
Soppet, Daniel R., Centreville, VA, United States
Lafleur, David W., Washington, DC, United States
Endress, Gregory A., Potomac, MD, United States
Ebner, Reinhard, Gaithersburg, MD, United States
Komatsoulis, George, Silver Spring, MD, United States
Duan, Roxanne D., Bethesda, MD, United States

PI US 2001021700 A1 20010913

AI US 2000-739254 A1 20001219 (9)

RLI-- Continuation of Ser. No. US 2000-511554, filed on 23 Feb 2000, ABANDONED
Continuation-in-part of Ser. No. WO 1999-US19330, filed on 24 Aug 1999,
UNKNOWN

PRAI US 1998-97917P 19980825 (60)

US 1998-98634P 19980831 (60)

DT Utility

FS APPLICATION

LREP HUMAN GENOME SCIENCES INC, 9410 KEY WEST AVENUE, ROCKVILLE, MD, 20850

CLMN Number of Claims: 23

ECL Exemplary Claim: 1

DRWN No Drawings

LN.CNT 15462

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The present invention relates to novel human secreted proteins and isolated nucleic acids containing the coding regions of the genes

encoding such proteins. Also provided are vectors, host cells, antibodies, and recombinant methods for producing human secreted proteins. The invention further relates to diagnostic and therapeutic methods useful for diagnosing and treating diseases, disorders, and/or conditions related to these novel human secreted proteins.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

SUMM . . . Clin. Invest. 79:1440-1446, 1987); anticollagenase-serum; alpha2-antiplasmin (Holmes et al., J. Biol. Chem. 262(4):1659-1664, 1987); Bisantrene (National Cancer Institute); Lobenzarit disodium (N-(2)-**carboxyphenyl**-4-chloroanthronilic acid disodium or "CCA"; Takeuchi et al., Agents Actions 36:312-316, 1992); Thalidomide; Angostatic steroid; AGM-1470; carboxynaminolmidazole; and metalloproteinase inhibitors such. . .

SUMM . . . that may be used according to the methods of the invention include, but are not limited to, glutamyl derivatives of **benzoic acid** mustard alkylating agent, phosphate derivatives of etoposide or mitomycin C, cytosine arabinoside, daunorubisin, and phenoxyacetamide derivatives of doxorubicin.

DETD . . . Three major families of cell surface antigens can be identified on monocytes: adhesion molecules, molecules involved in antigen presentation, and **Fc receptor**. Modulation of the expression of MHC class II antigens and other costimulatory molecules, such as B7 and ICAM-1, may result. . .

L8 ANSWER 39 OF 61 USPATFULL

AN 2000:28133 USPATFULL

TI Heterocyclic compounds, their preparation and their use as leucocyte adhesion inhibitors and VLA-4-antagonists

IN Wehner, Volkmar, Sandberg, Germany, Federal Republic of
Stilz, Hans Ulrich, Frankfurt, Germany, Federal Republic of
Schmidt, Wolfgang, Frankfurt, Germany, Federal Republic of

PA Hoechst Marion Roussel Deutschland GmbH, Frankfurt am Main, Germany,
Federal Republic of (non-U.S. corporation)

PI US 6034238 20000307

AI US 1998-158772 19980923 (9)

PRAI DE 1997-19741873 19970923

DT Utility

FS Granted

EXNAM Primary Examiner: Higel, Floyd D.

LREP Foley & Lardner

CLMN Number of Claims: 15

ECL Exemplary Claim: 1

DRWN No Drawings

LN.CNT 2807

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB Compounds of the formula I, ##STR1## in which B, E, W, Y, Z, R, R.sup.2, R.sup.2a, R.sup.2b, R.sup.3, g and h have the meanings indicated in the specifications. The compounds of the formula I are valuable pharmaceutical active compounds, which are suitable, for example, for the therapy and prophylaxis of inflammatory disorders, for example of rheumatoid arthritis, or of allergic disorders. The compounds of the formula I are inhibitors of the adhesion and migration of leucocytes and/or antagonists of the adhesion receptor VLA-4 belonging to the integrins group. They are generally suitable for the therapy or prophylaxis of illnesses which are caused by an undesired extent of leucocyte adhesion and/or leucocyte migration or are associated therewith, or in which cell-cell or cell-matrix interactions which are based on interactions of VLA-4 receptors with their ligands play a part. The invention furthermore relates to processes for the preparation of the compounds of the formula I, their use in the therapy and prophylaxis of the disease states mentioned and pharmaceutical preparations which contain the compounds of the formula I.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

SUMM . . . sulfuric acid, nitric acid, methanesulfonic acid,

p-toluenesulfonic acid, naphthalenedisulfonic acids, oxalic acid, acetic acid, tartaric acid, lactic acid, salicylic acid, **benzoic acid**, formic acid, propionic acid, pivalic acid, diethylacetic acid, malonic acid, succinic acid, pimelic acid, fumaric acid, maleic acid, malic acid, . . .

DETD 2.4 The plates were incubated at room temperature for 20 minutes with 100 .mu.l/well of **Fc receptor** blocking buffer (1 mg/ml of .gamma.-globulin, 100 mM NaCl, 100 .mu.M MgCl.sub.2, 100 .mu.M MnCl.sub.2, 100 .mu.M CaCl.sub.2, 1 mg/ml of BSA in 50 mM HEPES, pH 7.5). After removal of the **Fc receptor** blocking buffer washing was carried out once with PBS.

DETD 2.6 U937 cells were incubated in **Fc receptor** blocking buffer for 20 minutes and then added by pipette in a concentration of 1.times.10.sup.6 /ml and in an amount. . .

L8 ANSWER 40 OF 61 USPATFULL

AN 2000:7195 USPATFULL

TI Method for stimulating an immune response utilizing recombinant alphavirus particles

IN Dubensky, Jr., Thomas W., Rancho Sante Fe, CA, United States

Polo, John M., San Diego, CA, United States

Chang, Steven M.W., San Diego, CA, United States

Jolly, Douglas J., Leucadia, CA, United States

PA Chiron Corporation, Emeryville, CA, United States (U.S. corporation)

PI US 6015694 20000118

AI US 1997-931869 19970916 (8)

RLI Division of Ser. No. US 1995-404796, filed on 15 Mar 1995 which is a continuation-in-part of Ser. No. US 1995-376184, filed on 18 Jan 1995, now abandoned which is a continuation-in-part of Ser. No. US 1994-348472, filed on 30 Nov 1994, now abandoned which is a continuation-in-part of Ser. No. US 1994-198450, filed on 18 Feb 1994, now abandoned which is a continuation-in-part of Ser. No. US 1993-122791, filed on 15 Sep 1993, now abandoned

DT Utility

FS Granted

EXNAM Primary Examiner: Brusca, John S.

LREP McMasters, David D., Blackburn, Robert P.

CLMN Number of Claims: 11

ECL Exemplary Claim: 1

DRWN 35 Drawing Figure(s); 30 Drawing Page(s)

LN.CNT 10431

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The present invention provides compositions and methods for utilizing recombinant alphavirus vectors. Also disclosed are compositions and methods for making and utilizing eukaryotic layered vector initiation systems.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

DETD . . . 89:33, 1992); carboxypeptidase G2, which will cleave the glutamic acid from para-N-bis (2-chloroethyl) aminobenzoyl glutamic acid, thereby creating a toxic **benzoic acid** mustard; and Penicillin-V amidase, which will convert phenoxyacetabide derivatives of doxorubicin and melphalan to toxic compounds (see generally, Vrudhula et. . .

DETD . . . proteins that recognize Fc portions of antibodies. Monoclonal antibodies which recognize only preselected target cells are then bound to such **Fc receptor**-bearing alphavirus vector particles, such that the vector particles bind to and infect only those preselected target cells (for example, tumor. . .

L8 ANSWER 41 OF 61 USPATFULL

AN 2000:7187 USPATFULL

TI Eukaryotic layered vector initiation systems

IN Dubensky, Jr., Thomas W., Rancho Sante Fe, CA, United States

Polo, John M., San Diego, CA, United States

Jolly, Douglas J., Leucadia, CA, United States

Driver, David A., San Diego, CA, United States

PA Chiron Viagene, Inc., Emeryville, CA, United States (U.S. corporation)
PI US 6015686 20000118
AI US 1995-404796 19950315 (8)
RLI Continuation-in-part of Ser. No. US 1995-376184, filed on 20 Jan 1995,
now abandoned which is a continuation-in-part of Ser. No. US
1994-348472, filed on 30 Nov 1994, now abandoned which is a
continuation-in-part of Ser. No. US 1994-198450, filed on 18 Feb 1994,
now abandoned which is a continuation-in-part of Ser. No. US
1993-122791, filed on 15 Sep 1993, now abandoned
DT Utility
FS Granted
EXNAM Primary Examiner: Ketter, James; Assistant Examiner: Brusca, John S.
LREP Seed & Berry, Kruse, Norman J., Blackburn, Robert P.
CLMN Number of Claims: 20
ECL Exemplary Claim: 1
DRWN 37 Drawing Figure(s); 30 Drawing Page(s)
LN.CNT 10466

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The present invention provides compositions and methods for utilizing
recombinant alphavirus vectors. Also disclosed are compositions and
methods for making and utilizing eukaryotic layered vector initiation
systems.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

DETD . . . 89:33, 1992); carboxypeptidase G2, which will cleave the
glutamic acid from para-N-bis (2-chloroethyl) aminobenzoyl glutamic
acid, thereby creating a toxic **benzoic acid** mustard;
and Penicillin-V amidase, which will convert phenoxyacetabide
derivatives of doxorubicin and melphalan to toxic compounds (see
generally, Vrudhula et. . .

DETD . . . proteins that recognize Fc portions of antibodies. Monoclonal
antibodies which recognize only preselected target cells are then bound
to such **Fc receptor**-bearing alphavirus vector
particles, such that the vector particles bind to and infect only those
preselected target cells (for example, tumor. . .

L8 ANSWER 42 OF 61 USPATFULL

AN 1999:160063 USPATFULL

TI Heterocycles as inhibitors of leucocyte adhesion and as VLA-4
antagonists

IN Stilz, Hans Ulrich, Frankfurt, Germany, Federal Republic of
Wehner, Volkmar, Sandberg, Germany, Federal Republic of
Huels, Christoph, Wackernheim, Germany, Federal Republic of
Seiffge, Dirk, Mainz-Kostheim, Germany, Federal Republic of

PA Hoechst Aktiengesellschaft AG, Frankfurt, Germany, Federal Republic of
(non-U.S. corporation)

PI US 5998447 19991207

AI US 1997-972031 19971117 (8)

PRAI DE 1996-19647382 19961115

DT Utility

FS Granted

EXNAM Primary Examiner: Richter, Johann; Assistant Examiner: Keating, Dominic

LREP Foley & Lardner

CLMN Number of Claims: 17

ECL Exemplary Claim: 1

DRWN No Drawings

LN.CNT 4819

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB Compounds of the formula I ##STR1## in which B, D, E, R, W, Y, Z, b, c,
d, e, f, g and have the meanings indicated in the claims, are inhibitors
of the adhesion and migration of leucocytes and/or antagonists of the
adhesion receptor VLA-4 which belongs to the group of integrins. The
invention relates to the use of compounds of the formula I, and of
pharmaceutical preparations which contain such compounds, for the
treatment and prophylaxis of diseases which are caused by an un desired
extent of leucocyte adhesion and/or leucocyte migration or which are
associated therewith or in which cell--cell or cell-matrix interactions

play a part which are based on interactions of VLA-4 receptors with their ligands, for example of inflammatory processes, rheumatoid arthritis or allergic disorders, and it also relates to the use of compounds of the formula I for the production of pharmaceuticals for use in such diseases. It further relates to novel compounds of the formula I.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

SUMM . . . sulfuric acid or phosphoric acid, and with organic carboxylic or sulfonic acids, such as, for example, acetic acid, citric acid, **benzoic acid**, maleic acid, fumaric acid, tartaric acid, methanesulfonic acid or p-toluenesulfonic acid.

DETD 2.4 The plates were incubated at room temperature for 20 minutes with 100 .mu.l/well of **Fc receptor** blocking buffer (1 mg/ml of .gamma.-globulin, 100 mM NaCl, 100 .mu.M MgCl.sub.2, 100 .mu.M MnCl.sub.2, 100 .mu.M CaCl.sub.2, 1 mg/ml BSA in 50 mM HEPES, pH 7.5). After removing the **Fc receptor** blocking buffer, washing was carried out once with PBS.

DETD 2.6 U937 cells were incubated in **Fc receptor** blocking buffer for 20 minutes and then pipetted in at a concentration of 1.times.10.sup.6 /ml and in an amount of. . .

L8 ANSWER 43 OF 61 USPATFULL

AN 1999:141975 USPATFULL

TI Therapeutic inhibitor of vascular smooth muscle cells

IN Kunz, Lawrence L., Redmond, WA, United States

Klein, Richard A., Edmonds, WA, United States

Reno, John M., Brier, WA, United States

PA NeoRx Corporation, Seattle, WA, United States (U.S. corporation)

PI US 5981568 19991109

AI US 1997-829685 19970331 (8)

RLI Continuation-in-part of Ser. No. US 1995-450793, filed on 25 May 1995, now patented, Pat. No. US 5811447 which is a continuation of Ser. No. US 1993-62451, filed on 13 May 1993, now abandoned And a continuation-in-part of Ser. No. WO 1996-US2125, filed on 15 Feb 1996 which is a continuation-in-part of Ser. No. US 1995-389712, filed on 15 Feb 1995

DT Utility

FS Granted

EXNAM Primary Examiner: Barts, Samuel

LREP Schwegman, Lundberg, Woessner & Kluth, P.A.

CLMN Number of Claims: 56

ECL Exemplary Claim: 1

DRWN 30 Drawing Figure(s); 22 Drawing Page(s)

LN.CNT 5553

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB Methods are provided for inhibiting stenosis or restenosis following vascular trauma in a mammalian host, comprising administering to the host a therapeutically effective dosage of a cytostatic agent and/or cytoskeletal inhibitor so as to biologically stent the traumatized vessel. Also provided is a method to inhibit or reduce vascular remodeling following vascular trauma, comprising administering an effective amount of a cytoskeletal inhibitor. Further provided are pharmaceutical compositions and kits comprising the therapeutic agents of the invention.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

DETD . . . include inactive ingredients such as cellulose, pregelatinized starch, silicon dioxide, hydroxypropyl methylcellulose, magnesium stearate, microcrystalline cellulose, starch, talc, titanium dioxide, **benzoic acid**, citric acid, corn starch, mineral oil, polypropylene glycol, sodium phosphate, and zinc stearate, and the like. Hard or soft gelatin. . .

DETD . . . does not bind to sites in the patient through antigen-specific binding, but instead binds in a non-specific manner, e.g., through **Fc receptor** binding reticuloendothelial cells, asialo-receptor binding, and by binding to ubiquitin-expressing cells.

The irrelevant "blocker" decreases non-specific binding of the therapeutic. . .

L8 ANSWER 44 OF 61 USPATFULL
AN 1998:154038 USPATFULL
TI Methods of determining chemicals that modulate expression of genes associated with cardiovascular disease
IN Foulkes, J. Gordon, Huntington Station, NY, United States
Liechtfried, Franz E., Vienna, Austria
Pieler, Christian, Vienna, Austria
Stephenson, John R., Santa Cruz, CA, United States
Case, Casey C., Lynbrook, NY, United States
PA Oncogene Science, Inc., Uniondale, NY, United States (U.S. corporation)
PI US 5846720 19981208
AI US 1996-700757 19960815 (8)
RLI Continuation of Ser. No. US 1992-832905, filed on 7 Feb 1992, now patented, Pat. No. US 5580722 which is a continuation-in-part of Ser. No. US 1990-555196, filed on 18 Jul 1990, now abandoned which is a continuation-in-part of Ser. No. US 1989-382712, filed on 18 Jul 1989, now abandoned
DT Utility
FS Granted
EXNAM Primary Examiner: Zitomer, Stephanie W.
LREP White, John P. Cooper & Dunham LLP
CLMN Number of Claims: 7
ECL Exemplary Claim: 1
DRWN 47 Drawing Figure(s); 42 Drawing Page(s)
LN.CNT 3998

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The invention provided for a method of transcriptionally modulating the expression of a gene encoding a protein of interest associated with treatment of one or more symptoms of a cardiovascular disease. Further provided is a method of determining whether a molecule not previously known to be a modulator of protein biosynthesis is capable of directly and specifically transcriptionally modulating the expression of a gene encoding a protein of interest associated with treatment of one or more symptoms of a cardiovascular disease. Screening methods, including methods of essentially simultaneously screening molecules to determine whether the molecules are capable of directly and specifically transcriptionally modulating one or more genes encoding proteins of interest associated with treatment of one or more symptoms of a cardiovascular disease, are also provided.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

DETD . . . interest may be involved in the uptake of modified lipoproteins e.g. LDL-R, scavenger receptor, advanced glycosylated end-product receptor or macrophage **FC receptor**. The protein of interest may be involved in lipid metabolism e.g. AMP-activated protein kinase, AMP-activated protein kinase kinase, acetyl CoA. . .

DETD . . . pyrazine

1648 2-[5,6-Bis(4-sulfo-
0.86 7.69 1.00

phenyl)-1,2,4-triazine-
3-yl]-4-(4-sulfophenyl)-
pyridine, trisodium salt

1651 Bis(2,2,2-trifluoroethyl)
0.69 3.57 0.70

(methocarbonyl-methyl)-
phosphonate

1655 2,5-Bis(trifluoro-methyl)
0.54 4.76 0.81

benzoic acid

1703 3-Bromobenzonitrile
0.76 10.00

0.90

1704 4-Bromobenzonitrile
0.77 4.16 0.94

1705	4-Bromobenzophenone	0.54	14.28	0.62
1712	Calcein Blue	0.74	8.33	0.94
1720	(15)-(-)-Camphor	0.65	4.76	

L8 ANSWER 45 OF 61 USPATFULL
AN 1998:150739 USPATFULL
TI Alphavirus vector constructs
IN Dubensky, Jr., Thomas W., Rancho Sante Fe, CA, United States
Polo, John M., San Diego, CA, United States
Ibanez, Carlos E., San Diego, CA, United States
Chang, Stephen M. W., San Diego, CA, United States
Jolly, Douglas J., Leucadia, CA, United States
Driver, David A., San Diego, CA, United States
Belli, Barbara A., San Diego, CA, United States
PA Chiron Corporation, Emeryville, CA, United States (U.S. corporation)
PI US 5843723 19981201
AI US 1996-739167 19961030 (8)
RLI Continuation of Ser. No. US 1995-404796, filed on 20 Mar 1995 which is a continuation-in-part of Ser. No. US 1995-376184, filed on 20 Jan 1995, now abandoned which is a continuation-in-part of Ser. No. US 1994-348472, filed on 30 Nov 1994, now abandoned which is a continuation-in-part of Ser. No. US 1994-198450, filed on 18 Feb 1994, now abandoned which is a continuation-in-part of Ser. No. US 1993-122791, filed on 15 Sep 1993, now abandoned
DT Utility
FS Granted
EXNAM Primary Examiner: Ketter, James; Assistant Examiner: Brusca, John S.
LREP McMasters, David D., Kruse, Norman J., Blackburn, Robert P.
CLMN Number of Claims: 47
ECL Exemplary Claim: 1
DRWN 37 Drawing Figure(s); 30 Drawing Page(s)
LN.CNT 10318
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention provides compositions and method,, for utilizing recombinant alphavirus vectors.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

DETD . . . 89:33, 1992); carboxypeptidase G2, which will cleave the glutamic acid from para-N-bis (2-chloroethyl) aminobenzoyl glutamic acid, thereby creating a toxic **benzoic acid** mustard; and Penicillin-V amidase, which will convert phenoxyacetabide derivatives of doxorubicin and melphalan to toxic compounds (see generally, Vrudhula et. . .

DETD . . . proteins that recognize Fc portions of antibodies. Monoclonal antibodies which recognize only preselected target cells are then bound to such **Fc receptor**-bearing alphavirus vector particles, such that the vector particles bind to and infect only those preselected target cells (for example, tumor. . .

L8 ANSWER 46 OF 61 USPATFULL
AN 1998:119004 USPATFULL
TI Eukaryotic layered vector initiation systems
IN Dubensky, Jr., Thomas W., P.O. Box 675205, Rancho Sante Fe, CA, United States 92067
Polo, John M., 1222 Reed Ave., Number 4, San Diego, CA, United States 92109
Jolly, Douglas J., 277 Hillcrest Dr., Leucadia, CA, United States 92024
Driver, David A., 5142 Biltmore St., San Diego, CA, United States 92117
PI US 5814482 19980929
AI US 1996-739158 19961030 (8)
RLI Division of Ser. No. US 1995-404796, filed on 15 Mar 1995 which is a continuation-in-part of Ser. No. US 1995-376184, filed on 18 Jan 1995, now abandoned which is a continuation-in-part of Ser. No. US 1994-348472, filed on 30 Nov 1994, now abandoned which is a

continuation-in-part of Ser. No. US 1994-198450, filed on 18 Feb 1994, now abandoned which is a continuation-in-part of Ser. No. US 1993-122791, filed on 15 Sep 1993, now abandoned

DT Utility
FS Granted
EXNAM Primary Examiner: Ketter, James; Assistant Examiner: Brusca, John S.
LREP Seed & Berry, Kruse, Norman J., Blackburn, Robert P.
CLMN Number of Claims: 25
ECL Exemplary Claim: 1
DRWN 37 Drawing Figure(s); 30 Drawing Page(s)
LN.CNT 10431

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The present invention provides compositions and methods for utilizing recombinant alphavirus vectors. Also disclosed are compositions and methods for making and utilizing eukaryotic layered vector initiation systems.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

DETD . . . 89:33; 1992); carboxypeptidase G2, which will cleave the glutamic acid from para-N-bis (2-chloroethyl) aminobenzoyl glutamic acid, thereby creating a toxic **benzoic acid** mustard; and Penicillin-V amidase, which will convert phenoxyacetabide derivatives of doxorubicin and melphalan to toxic compounds (see generally, Vrudhula et. . .

DETD . . . proteins that recognize Fc portions of antibodies. Monoclonal antibodies which recognize only preselected target cells are then bound to such **Fc receptor**-bearing alphavirus vector particles, such that the vector particles bind to and infect only those preselected target cells (for example, tumor. . .

L8 ANSWER 47 OF 61 USPATFULL

AN 1998:91872 USPATFULL

TI Alphavirus structural protein expression cassettes
IN Dubensky, Jr., Thomas W., Rancho Sante Fe, CA, United States
Polo, John M., San Diego, CA, United States
Ibanez, Carlos E., San Diego, CA, United States
Chang, Stephen M. W., San Diego, CA, United States
Jolly, Douglas J., Leucadia, CA, United States
Driver, David A., San Diego, CA, United States

PA Chiron Corporation, Emeryville, CA, United States (U.S. corporation)

PI US 5789245 19980804

AI US 1996-741881 19961030 (8)

RLI Division of Ser. No. US 1995-404796, filed on 15 Mar 1995 which is a continuation-in-part of Ser. No. US 1995-376184, filed on 20 Jan 1995, now abandoned which is a continuation-in-part of Ser. No. US 1994-348472, filed on 30 Nov 1994, now abandoned which is a continuation-in-part of Ser. No. US 1994-198450, filed on 18 Feb 1994, now abandoned which is a continuation-in-part of Ser. No. US 1993-122791, filed on 15 Sep 1993, now abandoned

DT Utility

FS Granted

EXNAM Primary Examiner: Ketter, James; Assistant Examiner: Brusca, John S.

LREP McMasters, David D., Kruse, Norman J., Blackburn, Robert P.

CLMN Number of Claims: 29

ECL Exemplary Claim: 1

DRWN 35 Drawing Figure(s); 30 Drawing Page(s)

LN.CNT 10270

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The present invention provides compositions and methods for utilizing recombinant alphavirus vectors. Also disclosed are compositions and methods for making and utilizing eukaryotic layered vector initiation systems.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

DETD . . . 89:33, 1992); carboxypeptidase G2, which will cleave the glutamic acid from para-N-bis (2-chloroethyl) aminobenzoyl glutamic acid, thereby creating a toxic **benzoic acid** mustard;

and Penicillin-V amidase, which will convert phenoxyacetabide derivatives of doxorubicin and melphalan to toxic compounds (see generally, Vrudhula et. . . .

DETD . . . proteins that recognize Fc portions of antibodies. Monoclonal antibodies which recognize only preselected target cells are then bound to such **Fc receptor**-bearing alphavirus vector particles, such that the vector particles bind to and infect only those preselected target cells (for example, tumor. . . .

L8 ANSWER 48 OF 61 USPATFULL
AN 96:111313 USPATFULL
TI Methods of determining chemicals that modulate transcriptionally expression of genes associated with cardiovascular disease
IN Foulkes, J. Gordon, Huntington Station, NY, United States
Liechtfried, Franz E., Vienna, Austria
Pieler, Christian, Vienna, Austria
Stephenson, John R., Santa Cruz, CA, United States
Case, Casey C., Lynbrook, NY, United States
PA Oncogene Science, Inc., Uniondale, NY, United States (U.S. corporation)
PI US 5580722 19961203
AI US 1992-832905 19920207 (7)
RLI Continuation-in-part of Ser. No. US 1990-555196, filed on 18 Jul 1990, now abandoned which is a continuation-in-part of Ser. No. US 1989-382712, filed on 18 Jul 1989, now abandoned
DT Utility
FS Granted
EXNAM Primary Examiner: Zitomer, Stephanie W.
LREP White, John P.
CLMN Number of Claims: 7
ECL Exemplary Claim: 1
DRWN 46 Drawing Figure(s); 42 Drawing Page(s)
LN.CNT 4011

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The invention provided for a method of directly and specifically transcriptionally modulating the expression of a gene encoding a protein of interest associated with treatment of one or more symptoms of a cardiovascular disease such as atherosclerosis, restenosis or hypertension.

Further provided is a method of determining whether a molecule not previously known to be a modulator of protein biosynthesis is capable of directly and specifically transcriptionally modulating the expression of a gene encoding a protein of interest associated with treatment of one or more symptoms of a cardiovascular disease.

Lastly, the invention provides a method of directly and specifically transcriptionally modulating in a human being the expression of a gene encoding a protein of interest associated with treatment of one or more symptoms of a cardiovascular disease, thus ameliorating the disease.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

DETD . . . interest may be involved in the uptake of modified lipoproteins e.g. LDL-R, scavenger receptor, advanced glycosylated end-product receptor or macrophage **FC receptor**. The protein of interest may be involved in lipid metabolism e.g. AMP-activated protein kinase, AMP-activated protein kinase kinase, acetyl CoA. . . .

DETD . . . 5.55 0.60
1648 2-[5,6-Bis(4-sulfophenyl)-
0.86 7.69 1.00
1,2,4-triazine-4-yl]-4-(4-
sulfophenyl)-pyridine,
trisodium salt
1651 Bis(2,2,2-trifluoroethyl)
0.69 3.57 0.70
(methocarbonyl-methyl)-
phosphonate
1655 2,5-Bis(trifluoro-methyl)

		0.54	4.76	0.81
	benzoic acid			
1703	3-Bromobenzonitrile	0.76	10.00	
				0.90
1704	4-Bromobenzonitrile	0.77	4.16	0.94
1705	4-Bromobenzophenone	0.54	14.28	
				0.62
1712	Calcein Blue	0.74	8.33	0.94
1720	(15)-(-)-Camphor	0.65	4.76.	.

L8 ANSWER 49 OF 61 USPATFULL

AN 96:58355 USPATFULL

TI Benzothiophenes substituted at the 3-carbonyl

IN Carlson, Donald G., Indianapolis, IN, United States

Cullinan, George J., Trafalgar, IN, United States

Fahey, Kennan J., Indianapolis, IN, United States

Jackson, William T., Indianapolis, IN, United States

Roehm, Neal W., Zionsville, IN, United States

Spaethe, Stephen M., Carmel, IN, United States

PA Eli Lilly and Company, Indianapolis, IN, United States (U.S. corporation)

PI US 5532382 19960702

AI US 1995-402683 19950313 (8)

DT Utility

FS Granted

EXNAM Primary Examiner: Raymond, Richard L.; Assistant Examiner: Lambkin, Deborah

LREP Strode, Janelle D., Sales, James J., Boone, David E.

CLMN Number of Claims: 4

ECL Exemplary Claim: 1

DRWN No Drawings

LN.CNT 712

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB Provided are compounds of the formula II ##STR1## wherein R.sub.6 and R.sub.7 are independently hydrogen or C.sub.1 -C.sub.6 alkyl;

R.sub.5 is naphthyl, substituted naphthyl, or phenyl substituted one to three times with C.sub.1 -C.sub.6 alkoxy, C.sub.1 -C.sub.6 alkyl, phenyl, or hydroxy; with the proviso that if the phenyl is substituted once with hydroxy, it must be further be substituted once or twice with C.sub.1 -C.sub.6 alkoxy, C.sub.1 -C.sub.6 alkyl, phenyl or hydroxy, and pharmaceutically acceptable salts thereof.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

DETD To 100 mL of methylene chloride was added 2 drops DMF, 3 g (15 mmol) of 4-phenyl **benzoic acid**, and 15 mls of SOCl.sub.2. The mixture was heated to reflux for 16 hours then reduced to dryness. To the. . .

DETD

Ingredient	Quantity (mg/5 ml)
------------	--------------------

Active ingredient	0.1-1000	mg
Sodium carboxymethyl cellulose	50	mg
Syrup	1.25	mg
Benzoic acid solution	0.10	mL
Flavor	q.v.	
Color	q.v.	
Purified water to	5	mL

DETD . . . No. 45 mesh U.S. sieve and mixed with the sodium carboxymethyl cellulose and syrup to form a smooth paste. The **benzoic acid** solution, flavor, and color are diluted with some of the water and added, with stirring. Sufficient water is then added. . .

DETD . . . measures the production of LTC4 by the cytokine dependent mast cell line MCII, stimulated by cross linking the high affinity **Fc receptor** for IgE (Fc.sub..epsilon. R1).

L8 ANSWER 50 OF 61 USPATFULL

AN 96:38926 USPATFULL

TI Benzothiophenes to inhibit leukotrienes

IN Carlson, Donald G., Indianapolis, IN, United States

Cullinan, George J., Trafalgar, IN, United States

Fahey, Kennan J., Indianapolis, IN, United States

Jackson, William T., Indianapolis, IN, United States

Roehm, Neal W., Zionsville, IN, United States

Spaethe, Stephen M., Carmel, IN, United States

PA Eli Lilly and Company, Indianapolis, IN, United States (U.S. corporation)

PI US 5514704 19960507

AI US 1995-402598 19950313 (8)

DT Utility

FS Granted

EXNAM Primary Examiner: Raymond, Richard L.; Assistant Examiner: Lambkin, Deborah

LREP Strode, Janelle D., Sales, James J., Boone, David E.

CLMN Number of Claims: 6

ECL Exemplary Claim: 1

DRWN No Drawings

LN.CNT 714

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB A method of inhibiting leukotrienes comprising administering to a mammal in need thereof an effective amount of a compound having the formula ##STR1## wherein R.sub.1 and R.sub.2 are independently hydrogen or C.sub.1 -C.sub.6 alkyl;

R.sub.3 is hydrogen, or a group of the formula ##STR2## wherein R.sub.4 is phenyl, substituted phenyl, naphthyl or substituted naphthyl, with the proviso that when R.sub.1 and R.sub.2 are both C.sub.1 -C.sub.6 alkyl, R.sub.3 is not hydrogen; and pharmaceutically acceptable salts thereof.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

DETD To 100 mL of methylene chloride was added 2 drops DMF, 3 g (15 mmol) of 4-phenyl **benzoic acid**, and 15 mls of SOCl.sub.2. The mixture was heated to reflux for 16 hours then reduced to dryness. To the.

DETD

Ingredient	Quantity (mg/5 ml)	
Active ingredient	0.1-1000	mg
Sodium carboxymethyl cellulose	50	mg
Syrup	1.25	mg
Benzoic acid solution	0.10	mL
Flavor	q.v.	
Color	q.v.	
Purified water to	5	mL

DETD . . . No. 45 mesh U.S. sieve and mixed with the sodium carboxymethyl cellulose and syrup to form a smooth paste. The **benzoic acid** solution, flavor, and color are diluted with some of the water and added, with stirring. Sufficient water is then added.

DETD . . . measures the production of LTC4 by the cytokine dependent mast cell line MCII, stimulated by cross linking the high affinity **Fc receptor** for IgE (Fce.sub..epsilon. R1).

L8 ANSWER 51 OF 61 USPATFULL

AN 96:38925 USPATFULL

TI Benzothiophene compounds useful for inhibiting lipoxxygenase

IN Carlson, Donald G., Indianapolis, IN, United States
Cullinan, George J., Trafalgar, IN, United States
Fahey, Kennan J., Indianapolis, IN, United States
Jackson, William T., Indianapolis, IN, United States
Roehm, Neal W., Zionsville, IN, United States
Spaethe, Stephen M., Carmel, IN, United States
PA Eli Lilly and Company, Indianapolis, IN, United States (U.S.
corporation)
PI US 5514703 19960507
AI US 1995-402593 19950313 (8)
DT Utility
FS Granted
EXNAM Primary Examiner: Raymond, Richard L.; Assistant Examiner: Lambkin,
Deborah
LREP Strode, Janelle D., Sales, James J., Boone, David E.
CLMN Number of Claims: 5
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 700

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB A method of inhibiting 5-lipoxygenase comprising administering to a
mammal in need thereof an effective amount of a compound having the
formula ##STR1## wherein R.sub.1 and R.sub.2 are independently hydrogen
or C.sub.1 -C.sub.6 alkyl;

R.sub.3 is hydrogen, or a group of the formula ##STR2## wherein R.sub.4
phenyl, substituted phenyl, naphthyl or substituted naphthyl, with the
proviso that when R.sub.1 and R.sub.2 are both C.sub.1 -C.sub.6 alkyl,
R.sub.3 is not hydrogen; and pharmaceutically acceptable salts thereof.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

DETD To 100 mL of methylene chloride was added 2 drops DMF, 3 g (15 mmol) of
4-phenyl **benzoic acid**, and 15 mls of SOCl.sub.2. The
mixture was heated to reflux for 16 hours then reduced to dryness. To
the. . .

DETD

Formulation 8: Suspensions

Ingredient	Quantity (mg/5 ml)
Active ingredient	0.1-1000 mg
Sodium carboxymethyl cellulose	50 mg
Syrup	1.25 mg
Benzoic acid solution	0.10 mL
Flavor	q.v.
Color	q.v.
Purified water to	5 mL

DETD . . . No. 45 mesh U.S. sieve and mixed with the sodium carboxymethyl
cellulose and syrup to form a smooth paste. The **benzoic
acid** solution, flavor, and color are diluted with some of the
water and added, with stirring. Sufficient water is then added. . .

DETD . . . measures the production of LTC₄ by the cytokine dependent mast
cell line MCII, stimulated by cross linking the high affinity **Fc
receptor** for IgE (Fc.sub..epsilon. R1).

L8 ANSWER 52 OF 61 USPATFULL

AN 95:112346 USPATFULL

TI Cancerous B cell treatment using substituted nucleoside derivatives

IN Goodman, Michael G., Rancho Santa Fe, CA, United States

Piro, Lawrence D., La Jolla, CA, United States

PA The Scripps Research Institute, La Jolla, CA, United States (U.S.
corporation)

PI US 5476659 19951219

AI US 1993-151142 19931112 (8)

RLI Continuation-in-part of Ser. No. US 1992-975830, filed on 13 Nov 1992,

now abandoned which is a continuation-in-part of Ser. No. US 1992-945215, filed on 15 Sep 1992, now patented, Pat. No. US 5317013 which is a division of Ser. No. US 1990-562101, filed on 2 Aug 1990, now patented, Pat. No. US 5147636 which is a division of Ser. No. US 1989-361974, filed on 9 Jun 1989, now patented, Pat. No. US 4948730 which is a division of Ser. No. US 1987-14618, filed on 13 Feb 1987, now patented, Pat. No. US 4849411 which is a continuation of Ser. No. US 1983-546679, filed on 1 Nov 1983, now patented, Pat. No. US 4643992 which is a continuation-in-part of Ser. No. US 1982-439846, filed on 9 Nov 1982, now patented, Pat. No. US 4539205

DT Utility

FS Granted

EXNAM Primary Examiner: Kim, Kay K. A.

LREP Welsh & Katz, Ltd.

CLMN Number of Claims: 32

ECL Exemplary Claim: 1

DRWN 7 Drawing Figure(s); 7 Drawing Page(s)

LN.CNT 2109

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB Processes for the killing of cancerous B cells, and particularly chronic lymphocytic leukemia (CLL) cells are disclosed. In one process, cancerous B cells that do not proliferate when contacted with an immune response-enhancing agent are contacted with an amount of such an agent sufficient to cause peripheral CLL cells to undergo blast transformation and proliferation. The contacted cells are then maintained for a time period sufficient for them to die from that contact. Further contacting of those cells with a cytotoxic amount of an anti-cancer drug or cytotoxic conjugate enhances the death of those cancer cells. In another process, peripheral CLL cells that proliferate on contact with an immune response-enhancing-agent are contacted with a proliferation-inducing amount of such an agent. The contacted cells are maintained for a time period sufficient to undergo blast transformation and proliferation, and the blasts are then contacted with a cytotoxic amount of an anti-cancer drug or cytotoxic conjugate and maintained.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

SUMM R.sub.1 contains up to about 20 atoms and has a Hammett substituent sigma constant for ionization of a meta-substituted **benzoic acid** that is greater than that of hydrogen;

DETD . . . antigens in a dose-dependent manner. Included among those up-regulated antigens are the well-known antigens denominated CD-22, CD-23 (low affinity IgE **Fc receptor**), CD-25 (IL-2 receptor; p55, Tac), CD-38 and CD-54 (ICAM-1).

DETD With reference to Hammett substituent sigma constants for meta **benzoic acid** substituents, the preferred 8-substituents have positive values. More preferably, the 8-substituents have sigma constants of about 0.1 to about 0.7.. . .

CLM What is claimed is:

. . . ; R.sub.1 contains up to about 20 atoms and has a Hammett substituent sigma constant for ionization of a meta-substituted **benzoic acid** that is greater than that of hydrogen; R.sub.2 is a radical having a length up to about that of an. . . .

. . . ; R.sub.1 contains up to about 20 atoms and has a Hammett substituent sigma constant for ionization of a meta-substituted **benzoic acid** that is greater than that of hydrogen; R.sub.2 is a radical having a length up to about that of an. . . .

. . . ; R.sub.1 contains up to about 20 atoms and has a Hammett substituent sigma constant for ionization of a meta-substituted **benzoic acid** that is greater than that of hydrogen; R.sub.2 is a radical having a length up to about that of an. . . .

L8 ANSWER 53 OF 61 USPATFULL

AN 94:46970 USPATFULL

TI Modulation of animal cellular responses with compositions containing 8-substituted guanine derivatives

IN Goodman, Michael G., Carlsbad, CA, United States
Weigle, William O., Del Mar, CA, United States

PA Scripps Clinic and Research Foundation, La Jolla, CA, United States
(U.S. corporation)
PI US 5317013 19940531
AI US 1992-945215 19920915 (7)
RLI Division of Ser. No. US 1990-562101, filed on 2 Aug 1990, now patented,
Pat. No. US 5147636 which is a division of Ser. No. US 1989-361974,
filed on 6 Jun 1989, now patented, Pat. No. US 4948730 which is a
division of Ser. No. US 1987-14618, filed on 13 Feb 1987, now patented,
Pat. No. US 4849411 which is a continuation of Ser. No. US 1983-546679,
filed on 1 Nov 1983, now patented, Pat. No. US 4643992 which is a
continuation-in-part of Ser. No. US 1982-439846, filed on 9 Nov 1982,
now patented, Pat. No. US 4539205
DT Utility
FS Granted
EXNAM Primary Examiner: Hill, Jr., Robert J.; Assistant Examiner: Carlson, K.
Cochrane
LREP Welsh & Katz, Ltd.
CLMN Number of Claims: 9
ECL Exemplary Claim: 1
DRWN 38 Drawing Figure(s); 31 Drawing Page(s)
LN.CNT 2169

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB Compositions and methods for their use in modulating animal cellular
responses are disclosed. The compositions include as an active agent an
effective amount of an 8-substituted guanine derivative bonded 9-1' to
an aldose having 5 or 6 carbon atoms in the aldose chain. The
composition includes a diluent amount of a physiologically tolerable
carrier. The guanine derivative is free of electrically charged
functionality, while the 8-substituent has an electron withdrawing
inductive effect greater than that of hydrogen and contains fewer than
about 15 atoms. Animal cellular responses are modulated by contacting
the cells with a composition of this invention.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

DETD . . . Cells bone marrow-derived lymphocytes
8-BrcGMP 8-bromoguanosine 3',5'-cyclic
monophosphate
8-BrGuo 8-bromoguanosine
Con A concanavalin A
CR complement receptor
cGMP guanosine 3',5'-cyclic
monophosphate
EA erythrocyte-antibody complexes
EAC erythrocyte-antibody-complement
complexes
FcR **Fc receptor**
FCS fetal calf serum
Guo guanosine
8-HGuo 8-haloguanosine
[.sup.3 H]TdR tritium labelled
deoxyribosylthymidine
Ia antigen antigens controlled by the
immune response genes
IgA,D,E,G and M
immunoglobulins. . .
DETD With reference to Hammett substituent sigma constants for meta
benzoic acid substituents, the preferred
8-substituents have positive values. More preferably, the 8-substituents
have sigma constants of about 0.1 to about 0.7. . .
DETD . . . interface cells collected separately. Rosettes were lysed in
0.83% NH.sub.4 Cl, and cells were counted, washed, and used in culture.
Fc receptor-bearing cells were separated by EA
rosetting (Parish and Hayward, supra), following the above protocol,
with the omission of fresh mouse. . .

TI Modulation of animal cellular responses with compositions containing
 8-substituted guanine derivatives and interferons
 IN Goodman, Michael G., Carlsbad, CA, United States
 Weigle, William O., Del Mar, CA, United States
 PA Scripps Clinic and Research Foundation, La Jolla, CA, United States
 (U.S. corporation)
 PI US 5147636 19920915
 AI US 1990-562101 19900802 (7)
 RLI Division of Ser. No. US 1989-361974, filed on 6 Jun 1989, now patented,
 Pat. No. US 4948730 which is a division of Ser. No. US 1987-14618, filed
 on 13 Feb. 1987, now patented, Pat. No. US 4849411 which is a
 continuation of Ser. No. US 1983-546679, filed on 1 Nov 1983, now
 patented, Pat. No. US 4643992 which is a continuation-in-part of Ser.
 No. US 1982-439846, filed on 9 Nov 1982, now patented, Pat. No. US
 4539205
 DT Utility
 FS Granted
 EXNAM Primary Examiner: Wax, Robert A.; Assistant Examiner: Ekstrom, Richard
 LREP Dressler, Goldsmith, Shore, Sutker & Milnamow, Ltd.
 CLMN Number of Claims: 9
 ECL Exemplary Claim: 1
 DRWN 32 Drawing Figure(s); 31 Drawing Page(s)
 LN.CNT 2196

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB Compositions and methods for their use in modulating animal cellular
 responses are disclosed. The compositions include as an active agent an
 effective amount of an 8-substituted guanine derivative bonded 9-1' to an
 aldose having 5 or 6 carbon atoms in the aldose chain. The composition
 includes a diluent amount of a physiologically tolerable carrier. The
 guanine derivative is free of electrically charged functionality, while
 the 8-substituent has an electron withdrawing inductive effect greater
 than that of hydrogen and contains fewer than about 15 atoms. Animal
 cellular responses are modulated by contacting the cells with a
 composition of this invention.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

DETD . . . Cells bone marrow-derived lymphocytes
 8-BrcGMP 8-bromoguanosine 3',5'-cyclic
 monophosphate
 8-BrGuo 8-bromoguanosine
 Con A concanavalin A
 CR complement receptor
 cGMP guanosine 3',5'-cyclic
 monophosphate
 EA erythrocyte-antibody complexes
 EAC erythrocyte-antibody-complement
 complexes
 FcR **Fc receptor**
 FCS fetal calf serum
 Guo guanosine
 8-HGuo 8-haloguanosine
 [sup.3 H]Tdr tritium labelled
 deoxyribosylthymidine
 Ia antigen antigens controlled by the
 immune response genes
 IgA,D,E,G and M immunoglobulins. . .
 DETD With reference to Hammett substituent sigma constants for meta
benzoic acid substituents, the preferred
 8-substituents have positive values. More preferably, the 8-substituents
 have sigma constants of about 0.1 to about 0.7. . .
 DETD . . . interface cells collected separately. Rosettes were lysed in
 0.83% NH.sub.4 Cl, and cells were counted, washed, and used in culture.
Fc receptor-bearing cells were separated by EA
 rosetting (Parish and Hayward, supra), following the above protocol,
 with the omission of fresh mouse. . .

L8 ANSWER 55 OF 61 USPATFULL

AN 91:104199 USPATFULL

TI Methods of treating diseases characterized by interactions of IgG-containing immune complexes with macrophage Fc receptors using antiestrogenic benzothiophenes

IN Schreiber, Alan D., Philadelphia, PA, United States

PA University of Pennsylvania, Philadelphia, PA, United States (U.S. corporation)

PI US 5075321 19911224

AI US 1988-159714 19880224 (7)

RLI Continuation-in-part of Ser. No. US 1987-30028, filed on 24 Mar 1987, now abandoned And a continuation-in-part of Ser. No. US 1987-89790, filed on 27 Aug 1987, now patented, Pat. No. US 4902681

DT Utility

FS Granted

EXNAM Primary Examiner: Nutter, Nathan M.

LREP Woodcock Washburn Kurtz Mackiewicz & Norris

CLMN Number of Claims: 53

ECL Exemplary Claim: 1

DRWN 2 Drawing Figure(s); 2 Drawing Page(s)

LN.CNT 641

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB Clearance of antibody-coated cells from the circulation is modulated by administering an effective amount of certain benzothiophene derivatives, or the physiologically acceptable acid addition salts thereof. The compounds are useful in treating mammalian diseases characterized by interactions between IgG containing immune complexes and macrophage Fc receptors.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

SUMM In the spleen, IgG-containing immune complexes bind by the Fc region of IgG to macrophages at **Fc receptor** sites on the macrophage surface. The Fc portion of the immunoglobulin molecule (identified by papain cleavage) is believed to be.

DETD . . . metaphosphoric acid, succinic acid, formic acid, phthalic acid, lactic acid and the like, most preferably with hydrochloric acid, citric acid, **benzoic acid**, maleic acid, acetic and propionic acid. Although the free bases may be used, acid addition salts, such as keoxifene HCl, . . .

L8 ANSWER 56 OF 61 USPATFULL

AN 90:63453 USPATFULL

TI Modulation of animal cellular responses with compositions containing 8-substituted guanine derivatives

IN Goodman, Michael G., Carlsbad, CA, United States

PA Weigle, William O., Del Mar, CA, United States

PA Scripps Clinic and Research Foundation, La Jolla, CA, United States (U.S. corporation)

PI US 4948730 19900814

AI US 1989-361974 19890606 (7)

RLI Division of Ser. No. US 1987-14618, filed on 13 Feb 1987, now patented, Pat. No. US 4849411 which is a continuation of Ser. No. US 1983-546679, filed on 1 Nov 1983, now patented, Pat. No. US 4643992 which is a continuation-in-part of Ser. No. US 1982-439846, filed on 9 Nov 1982, now patented, Pat. No. US 4539205

DT Utility

FS Granted

EXNAM Primary Examiner: Hazel, Blondel

LREP Dressler, Goldsmith, Shore, Sutker & Milnamow, Ltd.

CLMN Number of Claims: 7

ECL Exemplary Claim: 1

DRWN 38 Drawing Figure(s); 31 Drawing Page(s)

LN.CNT 2159

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB Compositions and methods for their use in modulating animal cellular responses are disclosed. The compositions include as an active agent an

effective amount of an 8-substituted guanine derivative bonded 9-1' to an aldose having 5 or 6 carbon atoms in the aldose chain. The composition includes a diluent amount of a physiologically tolerable carrier. The guanine derivative is free of electrically charged functionality, while the 8-substituent has an electron withdrawing inductive effector greater than that of hydrogen and contains fewer than about 15 atoms. Animal cellular responses are modulated by contacting the cells with a composition of this invention.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

DETD . . . Cells bone marrow-derived lymphocytes
8-BrcGMP 8-bromoguanosine 3',5'-cyclic
monophosphate
8-BrGuo 8-bromoguanosine
Con A concanavalin A
CR complement receptor
cGMP guanosine 3',5'-cyclic
monophosphate
EA erythrocyte-antibody complexes
EAC erythrocyte-antibody-complement
complexes
FcR **Fc receptor**
FCS fetal calf serum
Guo guanosine
8-HGuo 8-haloguanosine
[.sup.3 H]TdR tritium labelled
deoxyribosylthymidine
Ia antigen antigens controlled by the
immune response genes
IgA,D,E,G and M immunoglobulins. . .
DETD With reference to Hammett substituent sigma constants for meta
benzoic acid substituents, the preferred
8-substituents have positive values. More preferably, the 8-substituents
have sigma constants of about 0.1 to about 0.7. . .
DETD . . . interface cells collected separately. Rosettes were lysed in
0.83% NH.sub.4 Cl, and cells were counted, washed, and used in culture.
Fc receptor-bearing cells were separated by EA
rosetting (Parish and Hayward, supra), following the above protocol,
with the omission of fresh mouse. . .

L8 ANSWER 57 OF 61 USPATFULL

AN 89:58721 USPATFULL

TI Modulation of animal cellular responses with compositions containing
8-substituted guanine derivatives

IN Goodman, Michael G., Carlsbad, CA, United States

Weigle, William O., Del Mar, CA, United States

PA Scripps Clinic and Research Foundation, La Jolla, CA, United States.
(U.S. corporation)

PI US 4849411 19890718

AI US 1987-14618 19870213 (7)

DCD 20020903

RLI Continuation of Ser. No. US 1983-546679, filed on 1 Nov 1983, now
patented, Pat. No. US 4643992 which is a continuation-in-part of Ser.
No. US 1982-439846, filed on 9 Nov 1982, now patented, Pat. No. US
4539205

DT Utility

FS Granted

EXNAM Primary Examiner: Hazel, Blondel

LREP Dressler, Goldsmith, Shore, Sutker & Milnamow, Ltd.

CLMN Number of Claims: 17

ECL Exemplary Claim: 1

DRWN 32 Drawing Figure(s); 31 Drawing Page(s)

LN.CNT 2206

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB Compositions and methods for their use in modulating animal cellular
responses are disclosed. The compositions include as an active agent an

effective amount of an 8-substituted guanine derivative bonded 9-1' to an aldose having 5 or 6 carbon atoms in the aldose chain. The composition includes a diluent amount of a physiologically tolerable carrier. The guanine derivative is free of electrically charged functionality, while the 8-substituent has an electron withdrawing inductive effect greater than that of hydrogen and contains fewer than about 15 atoms. Animal cellular responses are modulated by contacting the cells with a composition of this invention.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

DETD . . . Cells bone marrow-derived lymphocytes
8-BrcGMP 8-bromoguanosine 3',5'-cyclic
monophosphate
8-BrGuo 8-bromoguanosine
Con A concanavalin A
CR complement receptor
cGMP guanosine 3',5'-cyclic
monophosphate
EA erythrocyte-antibody complexes
EAC erythrocyte-antibody-complement
complexes
FCR **Fc receptor**
FCS fetal calf serum
Guo guanosine
8-HGuo 8-haloguanosine
[.sup.3 H]TdR tritium labelled
deoxyribosylthymidine
Ia antigen antigens controlled by the
immune response genes
IgA, D, E, G. . . .
DETD With reference to Hammett substituent sigma constants for meta
benzoic acid substituents, the preferred
8-substituents have positive values. More preferably, the 8-substituents
have sigma constants of about 0.1 to about 0.7.. . .
DETD . . . interface cells collected separately. Rosettes were lysed in
0.83% NH.sub.4 Cl, and cells were counted, washed, and used in culture.
Fc receptor-bearing cells were separated by EA
rosetting (Parish and Hayward, supra), following the above protocol,
with the omission of fresh mouse. . . .
CLM What is claimed is:
. . . guanine derivative being free of electrically charged functionality,
and said 8-substituent having a positive Hammett substituent sigma
constant for meta **benzoic acid** substituents and
containing fewer than 15 atoms, together with a diluent amount of a
physiologically tolerable carrier.

L8 ANSWER 58 OF 61 USPATFULL

AN 87:11415 USPATFULL

TI Modulation of animal cellular responses with compositions containing
8-substituted guanine derivatives

IN Goodman, Michael G., Carlsbad, CA, United States

Weigle, William O., Del Mar, CA, United States

PA Scripps Clinic and Research Foundation, La Jolla, CA, United States
(U.S. corporation)

PI US 4643992 19870217

AI US 1983-546679 19831101 (6)

RLI Continuation-in-part of Ser. No. US 1982-439846, filed on 9 Nov 1982,
now patented, Pat. No. US 4539205

DT Utility

FS Granted

EXNAM Primary Examiner: Hazel, Blondel

LREP Dressler, Goldsmith, Shore, Sutker & Milnamow Ltd.

CLMN Number of Claims: 4

ECL Exemplary Claim: 1

DRWN 32 Drawing Figure(s); 31 Drawing Page(s)

LN.CNT 2149

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB Compositions and methods for their use in modulating animal cellular responses are disclosed. The compositions include as an active agent an effective amount of an 8-substituted guanine derivative bonded 9-1' to an aldose having 5 or 6 carbon atoms in the aldose chain. The composition includes a diluent amount of a physiologically tolerable carrier. The guanine derivative is free of electrically charged functionality, while the 8-substituent has an electron withdrawing inductive effect greater than that of hydrogen and contains fewer than about 15 atoms. Animal cellular responses are modulated by contacting the cells with a composition of this invention.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

DETD . . . Cells bone marrow-derived lymphocytes
8-BrcGMP 8-bromoguanosine 3',5'-cyclic
monophosphate
8-BrGuo 8-bromoguanosine
Con A concanavalin A
CR complement receptor
CGMP guanosine 3',5'-cyclic
monophosphate
EA erythrocyte-antibody complexes
EAC erythrocyte-antibody-complement
complexes
FcR **Fc receptor**
FCS fetal calf serum
Guo guanosine
8-HGuo 8-haloguanosine
[.sup.3 H]TdR tritium labelled
deoxyribosylthymidine
Ia antigen antigens controlled by the
immune response genes
IgA,D,E,G and M
immunoglobulins.
DETD With reference to Hammett substituent sigma constants for meta
benzoic acid substituents, the preferred
8-substituents have positive values. More preferably, the 8-substituents
have sigma constants of about 0.1 to about 0.7..
DETD . . . interface cells collected separately. Rosettes were lysed in
0.83% NH.sub.4 Cl, and cells were counted, washed, and used in culture.
Fc receptor-bearing cells were separated by EA
rosetting (Parish and Hayward, supra), following the above protocol,
with the omission of fresh mouse.

L8 ANSWER 59 OF 61 USPATFULL

AN 85:56529 USPATFULL

TI Conjugate having cytotoxicity and process for the preparation thereof

IN Kato, Yoshinori, Hino, Japan
Umemoto, Naoji, Hino, Japan
Hara, Takeshi, Hachioji, Japan
Tsukada, Yutaka, Ebetsu, Japan
Hirai, Hidematsu, Sapporo, Japan

PA Teijin Limited, Osaka, Japan (non-U.S. corporation)

PI US 4543211 19850924

AI US 1983-563858 19831221 (6)

PRAI JP 1982-226237 19821224

DT Utility

FS Granted

EXNAM Primary Examiner: Schain, Howard E.

LREP Sughrue, Mion, Zinn, Macpeak, and Seas

CLMN Number of Claims: 11

ECL Exemplary Claim: 1

DRWN No Drawings

LN.CNT 987

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB A conjugate having cytotoxicity prepared by covalently binding a polymer which has cytotoxic substances linked to its side chains and a reactive

group at its terminal to an immunoglobulin, or its fragment, which is capable of selectively binding to a particular antigen possessed by cells to be killed.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

SUMM . . . the Fc part induces the indiscriminate adsorptive binding to cells other than target cells and also the binding to an **Fc receptor** on the cell membrane, thus reducing the capability of the conjugate having cytotoxicity to select cells to be killed. Furthermore, . . .

SUMM As concrete examples of the cross-linking agent expressed by formula (XIV), meta-(N-maleimido)**benzoic acid** ##STR27## meta-(N-maleimido)**benzoic acid** 2,4-dinitrophenylester, .beta.-(N-maleimido)propionic acid N-hydroxysuccinimide ester, etc. may be mentioned.

L8 ANSWER 60 OF 61 USPATFULL

AN 85:52187 USPATFULL

TI Modulation of animal cellular responses with compositions containing 8-substituted guanine derivatives

IN Goodman, Michael G., Carlsbad, CA, United States

Weigle, William O., Del Mar, CA, United States

PA Scripps Clinic and Research Foundation, La Jolla, CA, United States (U.S. corporation)

PI US 4539205 19850903

AI US 1982-439846 19821109 (6)

DT Utility

FS Granted

EXNAM Primary Examiner: Hazel, Blondel

LREP Dressler, Goldsmith, Shore, Sutker & Milnamow, Ltd.

CLMN Number of Claims: 20

ECL Exemplary Claim: 1

DRWN 28 Drawing Figure(s); 27 Drawing Page(s)

LN.CNT 1864

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB Compositions and methods for their use in modulating animal cellular responses are disclosed. The compositions include as an active agent an effective amount of an 8-substituted guanine derivative bonded 9-1' to an aldose having 5 or 6 carbon atoms in the aldose chain. The composition includes a diluent amount of a physiologically tolerable carrier. The guanine derivative is free of electrically charged functionality, while the 8-substituent has an electron withdrawing inductive effect greater than that of hydrogen and contains fewer than about 15 atoms. Animal cellular responses are modulated by contacting the cells with a composition of this invention.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

DRWD . . . Cells bone marrow-derived lymphocytes

8-BrCGMP 8-bromoguanosine 3',5'-cyclic monophosphate

8-BrGuo 8-bromoguanosine

Con A concanavalin A

CR complement receptor

cGMP guanosine 3',5'-cyclic monophosphate

EA erythrocyte-antibody complexes

EAC erythrocyte-antibody-complement complexes

FcR **Fc receptor**

FCS fetal calf serum

Guo guanosine

8-HGuo 8-haloguanosine

[.sup.3 H]TdR tritium labelled deoxyribosylthymidine

Ia antigen antigens controlled by the immune response genes

IgA, D, E, G. . .

DRWD With reference to Hammett substituent sigma constants for meta **benzoic acid** substituent, the preferred 8-substituents have positive values. More preferably, the 8-substituents have sigma constants of about 0.1 to about 0.7.. . .

DRWD . . . interface cells collected separately. Rosettes were lysed in 0.83% NH.sub.4 Cl, and cells were counted, washed, and used in culture. **Fc receptor**-bearing cells were separated by EA rosetting (Parish and Hayward, supra), following the above protocol, with the omission of fresh mouse. . . .

L8 ANSWER 61 OF 61 USPATFULL
AN 85:17723 USPATFULL
TI Conjugate having cytotoxicity and process for the preparation thereof
IN Kato, Yoshinori, Hino, Japan
Umamoto, Naoji, Hino, Japan
Saito, Masahiko, Saitama, Japan
Hara, Takeshi, Hachioji, Japan
PA Teijin Limited, Osaka, Japan (non-U.S. corporation)
PI US 4507234 19850326
AI US 1983-563860 19831221 (6)
PRAI JP 1982-226236 19821224
DT Utility
FS Granted
EXNAM Primary Examiner: Schain, Howard E.
LREP Sughrue, Mion, Zinn, Macpeak and Seas
CLMN Number of Claims: 10
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 1213

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB A conjugate having cytotoxicity prepared by covalently binding a serum albumin having cytotoxic substance linked thereto to an immunoglobulin, or its fragment, which is able to bind selectively with a particular antigen of cells to be killed.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

SUMM . . . it, the Fc part induces the indiscriminate adsorptive binding to cells other than target cells and also the binding to **Fc receptor** on the cell membrane, thus reducing the capability of the conjugate having cytotoxicity to select cells to be killed. Furthermore, . . .

SUMM As concrete examples of the cross-linking agent expressed by formula (XIV), meta-(N-maleimido)**benzoic acid** N-hydroxysuccinimide ester ##STR25## meta-(N-maleimido)**benzoic acid** 2,4-dinitrophenylester, .beta.-(N-maleimido)propionic acid N-hydroxysuccinimido ester, etc. may be mentioned.

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